

*The Alan E. Lindsay*



# ECG Learning Center

*In Cyberspace*

**Dr. Alan  
Lindsay:**

"A teacher  
of substance  
and style"



**Whats New:** [Advanced ECG Quiz](#)  
(Requires Internet)



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# THE ALAN E. LINDSAY ECG LEARNING CENTER

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This tutorial is dedicated to the memory of Dr. Alan E. Lindsay, master teacher of electrocardiography, friend, mentor, and colleague. Many of the excellent ECG tracings illustrated in this learning program are from Dr. Lindsay's personal collection of ECG treasures. For many years these ECG's have been used in the training of medical students, nurses, housestaff physicians, cardiology fellows, and practicing physicians in Salt Lake City, Utah as well as at many regional and national medical meetings. It is an honor to be able to provide this tutorial on the World Wide Web in recognition of Dr. Lindsay's great love for teaching and for electrocardiography.

This interactive ECG tutorial represents an introduction to clinical electrocardiography. ECG terminology and diagnostic criteria often vary from book to book and from one teacher to another. In this tutorial an attempt has been made to conform to standardized terminology and criteria, although new diagnostic concepts derived from the recent ECG literature have been included in some of the sections. Finally, it is important to recognize that the mastery of ECG interpretation, one of the most useful clinical tools in medicine, can only occur if one acquires considerable experience in reading ECG's and correlating the specific ECG findings with the pathophysiology and clinical status of the patient.

The tutorial is organized in sections based on a recommended ["Method" of ECG interpretation](#). Each section provides some didactic teaching points, often linked to illustrations, and an interactive quiz. Beginning students should first go through the sections in the order in which they are presented. Others may chose to explore topics of interest in any order they wish. The ECG's range from the



sublime to the ridiculous, from simplicity to complexity, and from boring to fascinating. It is hoped that students will be left with some of the love of electrocardiography shared by Dr. Lindsay.

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## ECG Image Index

The following ECG categories contain hundreds of ECGs that range from the sublime to the ridiculous, from simplicity to complexity, and from boring to fascinating. Many of the ECG rhythm strips come from the collection of the late Dr. Alan Lindsay, master teacher of electrocardiography. Most of the 12- and 6-lead ECGs were recorded at LDS Hospital in Salt Lake City, Utah. Marquette Electronics has also given permission to use ECG rhythms and diagrams from their educational posters. Each of the ECGs has an interpretation and many have additional explanations that help explain the diagnosis. Feedback is encouraged using the feedback form provided with this website.

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[ecg\\_12lead056.gif](#)--Long QT: An ECG Marker For Sudden Cardiac Death

[ecg\\_12lead060.gif](#)--Hyperkalemia and Old Inferior MI

[ecg\\_12lead061.gif](#)--Advanced Hyperkalemia

[ecg\\_486.gif](#)--Giant TU Fusion Waves

[ecg\\_6lead018.gif](#)--Hypothermia: J-waves or Osborne Waves

## 13. Odds & Ends

[ecg\\_12lead001.gif](#)--Lead Error: V1 & V3 are Transposed

[ecg\\_12lead001z.gif](#)--Lead Error: V1 and V3 are Transposed!

[ecg\\_ac.gif](#)--60 Cycle Artifact - Marquette

[ecg\\_baseline.gif](#)--Wandering Baseline Artifact - Marquette

[ecg\\_calibration.gif](#)--Calibration Signal - Marquette

[ecg\\_tremor.gif](#)--Muscle Tremor Artifact - Marquette





## ECG Quizzes Lessons 1-12

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**Basic Quizzes** To access these quizzes, you must be connected to the Internet

1. [The 12 Lead ECG and Method of Interpretation \(Lessons I and II\)](#)
2. [Normal ECG Characteristics and Measurement Abnormalities \(Lessons III and IV\)](#)
3. [Arrhythmias \(Lesson V\)](#)
4. [Conduction Abnormalities \(Lesson VI\)](#)
5. [Atrial Enlargement and Ventricular Hypertrophy \(Lessons VII and VIII\)](#)
6. [Myocardial Infarctions \(Lesson IX\)](#)
7. [ST, T, and U Waves \(Lessons X, XI, and XII\)](#)

### Advanced Quizzes

1. [Quiz 1](#)





## ACC/AHA Clinical Competence in ECG Diagnoses

The following list of ECG diagnoses was derived from a recently published statement of the American College of Cardiology/American Heart Association (ACC/AHA) Committee to Develop a Clinical Competence Statement on Electrocardiography and Ambulatory Electrocardiography (J Am Coll Cardiol 2001;38:2091-2100). These diagnoses are considered to be the minimum knowledge necessary for competence in interpreting 12-lead ECGs. Items in the list are linked to topics in the [ECG Outline](#) of the Alan E. Lindsay ECG Learning Center or to ECG examples in the [Image Index](#).

### Normal Tracing

- [Normal ECG](#)

### Technical Problem

- Lead Misplaced
- Artifact
  - [Normal Variants or Artifacts](#)
  - [60 Cycle Artifact](#)
  - [Wandering Baseline Artifact](#)
  - [Muscle Tremor Artifact](#)

### Sinus Rhythms/Arrhythmias

- Sinus rhythm (50-90 bpm)
- Sinus tachycardia (>90 bpm)
- [Sinus bradycardia \(<50 bpm\)](#)
- [Sinus Arrhythmia](#)
- [Sinus arrest or pause](#)
- [Sino-atrial exit block](#)

### Other SV Arrhythmias

- [PAC's \(nonconducted\)](#)
- [PAC's \(conducted normally\)](#)
- [PAC's \(conducted with aberration\)](#)
- [Ectopic atrial rhythm or tachycardia \(unifocal\)](#)
- [Multifocal atrial rhythm or tachycardia](#)
- [Atrial fibrillation](#)
- Atrial flutter
  - [Atrial Flutter With 2:1 AV Conduction](#)
  - [Atrial Flutter With 2:1 AV Conduction](#)

- [Atrial Flutter With 3:2 AV Conduction](#)
- [Atrial Flutter with 3:2 Conduction Ratio](#)
- [Atrial Flutter With Variable AV Block And Rate-Dependent LBBB](#)
- [Atrial Flutter With 2:1 AV Conduction](#)
- [LBBB and Atrial Flutter with 2:1 AV Block](#)
- [Atrial Flutter With 2:1 and 4:1 Conduction and Rate Dependent LBBB](#)
- [Atrial Flutter With 2:1 AV Conduction](#)
- [Atrial Flutter With 2:1 AV Conduction](#)
- [Atrial Flutter With Variable AV Block](#)
- [Atrial Flutter With 2:1 Conduction](#)
- [Atrial Flutter with 2:1 Block](#)
- [Junctional prematures](#)
- [Junctional escapes or rhythms](#)
- [Accelerated Junctional rhythms](#)
- [Junctional tachycardia](#)
  - [Junctional Tachycardia With Exit Block](#)
  - [Junctional Tachycardia With and Without AV Block](#)
  - [Junctional Tachycardia With and Without Exit Block](#)
- [Paroxysmal supraventricular tachycardia](#)

## Ventricular Arrhythmias

- [PVC's](#)
- [Ventricular escapes or rhythm](#)
- [Accelerated ventricular rhythm](#)
- [Ventricular tachycardia \(uniform\)](#)
- [Ventricular tachycardia \(polymorphous or torsade\)](#)
- [Ventricular fibrillation](#)

## AV Conduction

- [1st degree AV block](#)
  - [1st Degree AV Block](#)
  - [A Most Unusual 1st Degree AV Block](#)
  - [Left Atrial Abnormality & 1st degree AV Block](#)
  - [Left Atrial Abnormality & 1st Degree AV Block](#)
  - [A Very Subtle 1st Degree AV Block](#)
- [Type I 2nd degree AV block \(Wenckebach\)](#)
- [Type II 2nd degree AV block \(Mobitz\)](#)
- [AV block, advanced \(high grade\)](#)
- [3rd degree AV block \(junctional escape rhythm\)](#)
- [3rd degree AV block \(ventricular escape rhythm\)](#)
- [AV dissociation \(default\)](#)
  - [Subsidiary escape pacemaker takes over by default](#)
- [AV dissociation \(usurpation\)](#)
  - [Incomplete AV Dissociation due to accelerated ventricular rhythm](#)

## Intraventricular Conduction

- [Complete LBBB, fixed or intermittent](#)
- [Incomplete LBBB](#)

- [Left anterior fascicular block \(LAFB\)](#)
- [Left posterior fascicular block \(LPFB\)](#)
- [Nonspecific IVCD](#)
- [WPW preexcitation pattern](#)

## QRS Axis and Voltage

- [Right axis deviation \(+90 to +180\)](#)
- [Left axis deviation \(-30 to -90\)](#)
- [Bizarre axis \(-90 to -180\)](#)
- [Indeterminate axis](#)
- [Low voltage frontal plane \(<0.5 mV\)](#)
- [Low voltage precordial \(<1.0 mV\)](#)

## Hypertrophy/Enlargements

- [Left atrial enlargement](#)
- [Right atrial enlargement](#)
- [Left ventricular hypertrophy](#)
- [Right ventricular hypertrophy](#)

## ST-T, and U Abnormalities

- [Early repolarization \(normal variant\)](#)
- [Nonspecific ST-T abnormalities](#)
  - [Left Atrial Enlargement and Nonspecific ST-T Wave Abnormalities](#)
  - [ST Segment Depression](#)
- [ST elevation \(transmural injury\)](#)
- [ST elevation \(pericarditis pattern\)](#)
- [Symmetrical T wave inversion](#)
  - [Inferior MI: Fully Evolved](#)
- [Hyperacute T waves](#)
- [Prominent upright U waves](#)
- [U wave inversion](#)
- [Prolonged QT interval](#)

## MI Patterns (acute, recent, old)

- [Inferior MI](#)
- [Inferoposterior MI](#)
- [Inferoposterolateral MI](#)
- [True posterior MI](#)
- [Anteroseptal MI](#)
- [Anterior MI](#)
- [Anterolateral MI](#)
- [High lateral MI](#)
- [Non Q-wave MI](#)
- [Right ventricular MI](#)

## Clinical Disorders

- Chronic pulmonary disease pattern
- [Suggests hypokalemia](#)
  - [Giant TU Fusion Waves](#)
- [Suggests hyperkalemia](#)
- Suggests hypocalcemia
- Suggests hypercalcemia
- [Suggests digoxin effect](#)





## The Alan E. Lindsay ECG Learning Center Feedback Form

Please let us know how we're doing!

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How useful is the online Electrocardiography information?

☐ Vital   ☐ Useful   ☐ Not Useful

Check all that apply:

☐ Patient   ☐ Student   ☐ Resident   ☐ Physician

☐ Other: (fill in)

Email address:

(please provide your name and e-mail address if you would like a reply)

Comments:

**Thank you for your input!**





## III. Characteristics of the Normal ECG

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I II **III** IV V VI VII VIII IX X XI XII

It is important to remember that there is a wide range of normal variability in the 12 lead ECG. The following "normal" ECG characteristics, therefore, are not absolute. It takes considerable ECG reading experience to discover all the normal variants. Only by following a structured ["Method of ECG Interpretation" \(Lesson II\)](#) and correlating the various ECG findings with the particular patient's clinical status will the ECG become a valuable clinical tool.

Topics for Study:

1. [Measurements](#)
2. [Rhythm](#)
3. [Conduction](#)
4. [Waveform description](#)

### 1. Measurements

➤ Heart Rate: 60 - 90 bpm

● [How to calculate the heart rate on ECG paper](#)

➤ PR Interval: 0.12 - 0.20 sec

➤ QRS Duration: 0.06 - 0.10 sec

➤ QT Interval ( $QT_c \leq 0.40$  sec)

● **Bazett's Formula:**  $QT_c = (QT)/\text{SqRoot } RR$  (in seconds)

● **Poor Man's Guide** to upper limits of QT: For HR = 70 bpm,  $QT \leq 0.40$  sec; for every 10 bpm increase above 70 subtract 0.02 sec, and for every 10 bpm decrease below 70 add 0.02 sec. For example:

■  $QT \leq 0.38$  @ 80 bpm

■  $QT \leq 0.42$  @ 60 bpm

➤ Frontal Plane QRS Axis:  $+90^\circ$  to  $-30^\circ$  (in the adult)

## 2. Rhythm:

### Normal sinus rhythm

The P waves in leads I and II must be upright (positive) if the rhythm is coming from the sinus node.

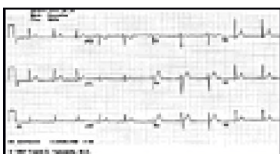
## 3. Conduction:

### Normal Sino-atrial (SA), Atrio-ventricular (AV), and Intraventricular (IV) conduction

Both the PR interval and QRS duration should be within the limits specified above.

## 4. Waveform Description:

(Normal ECG is shown below - Compare its waveforms to the descriptions below)



[Click to view](#)

## ➤ P Wave

It is important to remember that the P wave represents the *sequential* activation of the right and left atria, and it is common to see notched or biphasic P waves of right and left atrial activation.

- P duration < 0.12 sec
- P amplitude < 2.5 mm
- Frontal plane P wave axis: 0° to +75°
- May see notched P waves in frontal plane

## ➤ QRS Complex

The QRS represents the *simultaneous* activation of the right and left ventricles, although most of the QRS waveform is derived from the larger left ventricular musculature.

- QRS duration  $\leq$  0.10 sec
- QRS amplitude is quite variable from lead to lead and from person to person. Two determinates of QRS voltages are:

- Size of the ventricular chambers (i.e., the larger the chamber, the larger the voltage)

- Proximity of chest electrodes to ventricular chamber (the closer, the larger the voltage)

- Frontal plane leads:

- The normal QRS axis range (+90° to -30°); this implies that the QRS be mostly positive (upright) in leads II and I.

- Normal q-waves reflect normal septal activation (beginning on the LV septum); they are narrow (<0.04s duration) and small (<25% the amplitude of the R wave). They are often seen in leads I and aVL when the QRS axis is to the **left** of +60°, and in leads II, III, aVF when the QRS axis is to the **right** of +60°. Septal q waves should not be confused with the pathologic Q waves of myocardial infarction.

### ● Precordial leads: (see [Normal ECG](#))

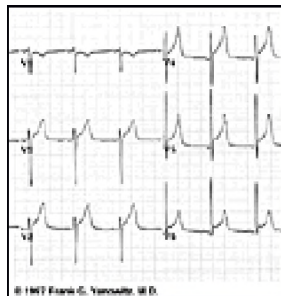
- Small r-waves begin in V1 or V2 and progress in size to V5. The R-V6 is usually smaller than R-V5.
- In reverse, the s-waves begin in V6 or V5 and progress in size to V2. S-V1 is usually smaller than S-V2.
- The usual transition from S>R in the right precordial leads to R>S in the left precordial leads is V3 or V4.
- Small "septal" q-waves may be seen in leads V5 and V6.

### ➤ ST Segment and T wave

In a sense, the term "ST segment" is a misnomer, because a discrete ST segment distinct from the T wave is usually absent. More often the ST-T wave is a smooth, continuous waveform beginning with the J-point (end of QRS), slowly rising to the peak of the T and followed by a rapid descent to the isoelectric baseline or the onset of the U wave. This gives rise to an asymmetrical T wave. In some normal individuals, particularly women, the T wave is symmetrical and a distinct, horizontal ST segment is present.

The normal T wave is usually in the same direction as the QRS except in the right precordial leads. In the normal ECG the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR.

● Normal ST segment elevation: this occurs in leads with large S waves (e.g., V1-3), and the normal configuration is **concave upward**. ST segment elevation with concave upward appearance may also be seen in other leads; this is often called **early repolarization**, although it's a term with little physiologic meaning (see example of "early repolarization" in leads V4-6):



[Click to view](#)

● **Convex or straight upward** ST segment elevation (e.g., leads II, III, aVF) is abnormal and suggests transmural injury or infarction:



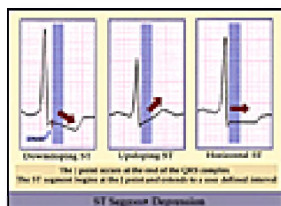
[Click to view](#)

● ST segment depression is always an abnormal finding, although often nonspecific (see ECG below):



[Click to view](#)

● ST segment depression is often characterized as "upsloping", "horizontal", or "downsloping".



[Click to view](#)

➤ The normal U Wave: (the most neglected of the ECG waveforms)

- U wave amplitude is usually  $< 1/3$  T wave amplitude in same lead
- U wave direction is the same as T wave direction in that lead
- U waves are more prominent at slow heart rates and usually best seen in the right precordial leads.
- Origin of the U wave is thought to be related to *afterdepolarizations* which interrupt or follow repolarization.









# I. The Standard 12 Lead ECG

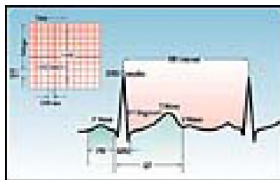
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I II III IV V VI VII VIII IX X XI XII

The standard 12-lead electrocardiogram is a representation of the heart's electrical activity recorded from electrodes on the body surface. This section describes the basic components of the ECG and the lead system used to record the ECG tracings.

Topics for study:

1. [ECG Waves and Intervals](#)
2. [Spatial Orientation of the 12 Lead ECG](#)



[click to view](#)

This diagram illustrates ECG waves and intervals as well as standard time and voltage measures on the ECG paper.

## 1. ECG Waves and Intervals:

What do they mean?

- P wave: the *sequential* activation (depolarization) of the right and left atria
- QRS complex: right and left ventricular depolarization (normally the ventricles are activated *simultaneously*)
- ST-T wave: ventricular repolarization

- U wave: origin for this wave is not clear - but probably represents "afterdepolarizations" in the ventricles
- PR interval: time interval from onset of atrial depolarization (P wave) to onset of ventricular depolarization (QRS complex)
- QRS duration: duration of ventricular muscle depolarization
- QT interval: duration of ventricular depolarization and repolarization
- RR interval: duration of ventricular cardiac cycle (an indicator of ventricular rate)
- PP interval: duration of atrial cycle (an indicator of atrial rate)

## 2. Orientation of the 12 Lead ECG

It is important to remember that the 12-lead ECG provides spatial information about the heart's electrical activity in 3 approximately orthogonal directions:

- Right ↔ Left
- Superior ↔ Inferior
- Anterior ↔ Posterior

Each of the 12 leads represents a particular orientation in space, as indicated below (RA = right arm; LA = left arm, LF = left foot):

- Bipolar limb leads (frontal plane):

- Lead I: RA (-) to LA (+) (Right Left, or lateral)
- Lead II: RA (-) to LF (+) (Superior Inferior)
- Lead III: LA (-) to LF (+) (Superior Inferior)

- Augmented unipolar limb leads (frontal plane):

- Lead aVR: RA (+) to [LA & LF] (-) (Rightward)
- Lead aVL: LA (+) to [RA & LF] (-) (Leftward)
- Lead aVF: LF (+) to [RA & LA] (-) (Inferior)

➤ Unipolar (+) chest leads (horizontal plane):

● Leads V1, V2, V3: (Posterior Anterior)

● Leads V4, V5, V6: (Right Left, or lateral)

Click here to see: [Lead Placement Diagrams](#) (*Requires an Internet connection*)





## II. A "Method" of ECG Interpretation

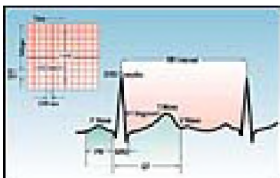
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I II III IV V VI VII VIII IX X XI XII

This "method" is recommended when reading all 12-lead ECG's. Like the physical examination, it is desirable to follow a standardized sequence of steps in order to avoid missing subtle abnormalities in the ECG tracing, some of which may have clinical importance. The 6 major sections in the "method" should be considered in the following order:

1. [Measurements](#)
2. [Rhythm Analysis](#)
3. [Conduction Analysis](#)
4. [Waveform Description](#)
5. [Ecg Interpretation](#)
6. [Comparison with Previous ECG \(if any\)](#)

### 1. Measurements (usually made in frontal plane leads):



[Click to view](#)

- Heart rate (state atrial and ventricular, if different)
- PR interval (from beginning of P to beginning of QRS)
- QRS duration (width of most representative QRS)

- QT interval (from beginning of QRS to end of T)
- QRS axis in frontal plane (go to: ["How To Determine Axis"](#))

Go to: [ECG Measurement Abnormalities \(Lesson IV\)](#) for description of normal and abnormal measurements

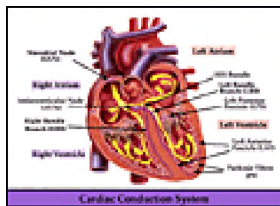
## 2. Rhythm Analysis

- State basic rhythm (e.g., "normal sinus rhythm", "atrial fibrillation", etc.)
- Identify additional rhythm events if present (e.g., "PVC's", "PAC's", etc)
- Consider all rhythm events from atria, AV junction, and ventricles

Go to: [ECG Rhythm Abnormalities \(Lesson V\)](#) for description of arrhythmias

## 3. Conduction Analysis

- "Normal" conduction implies normal sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction.



[Click to view](#)

The diagram illustrates the normal cardiac conduction system.

- The following conduction abnormalities are to be identified if present:
  - [SA block \(lesson VI\)](#): 2nd degree (type I vs. type II)
  - [AV block \(lesson VI\)](#): 1st, 2nd (type I vs. type II), and 3rd degree
  - [IV blocks \(lesson VI\)](#): bundle branch, fascicular, and nonspecific blocks
  - Exit blocks: blocks just distal to ectopic pacemaker site

(Go to ECG [Conduction Abnormalities \(Lesson VI\)](#) for a description of conduction abnormalities)

## 4. Waveform Description

➤ Carefully analyze the 12-lead ECG for abnormalities in each of the waveforms in the order in which they appear: P-waves, QRS complexes, ST segments, T waves, and... Don't forget the U waves.

● [P waves \(lesson VII\)](#): are they too wide, too tall, look funny (i.e., are they ectopic), etc.?

● QRS complexes: look for [pathologic Q waves \(lesson IX\)](#), [abnormal voltage \(lesson VIII\)](#), etc.

● [ST segments \(lesson X\)](#): look for abnormal ST elevation and/or depression.

● [T waves \(lesson XI\)](#): look for abnormally inverted T waves.

● [U waves \(lesson XII\)](#): look for prominent or inverted U waves.

## 5. ECG Interpretation

➤ This is the conclusion of the above analyses. Interpret the ECG as "Normal", or "Abnormal". Occasionally the term "borderline" is used if unsure about the significance of certain findings. List all abnormalities. Examples of "abnormal" statements are:

- Inferior MI, probably acute
- Old anteroseptal MI
- Left anterior fascicular block (LAFB)
- Left ventricular hypertrophy (LVH)
- Nonspecific ST-T wave abnormalities
- Any rhythm abnormalities

➤ Example:



[Click to view](#)

## 6. Comparison with previous ecg

➤ If there is a previous ECG in the patient's file, the current ECG should be compared with it to see if any significant changes have occurred. These changes may have important implications for clinical management decisions.



[Test your knowledge on lessons I and II by clicking here](#) (Internet connection required)



## IV. Abnormalities in the ECG Measurements

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I II III IV V VI VII VIII IX X XI XII

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Click on the measurement abnormality you would like to study

1. [Heart Rate](#)
2. [PR Interval](#)
3. [QRS Duration](#)
4. [QT Interval](#)
5. [QRS Axis](#)

### 1. Heart Rate

In normal sinus rhythm, a resting heart rate of below 60 bpm is called bradycardia and a rate of above 90 bpm is called tachycardia.

### 2. PR Interval

(measured from beginning of P to beginning of QRS in the frontal plane)

➤ Normal: 0.12 - 0.20s

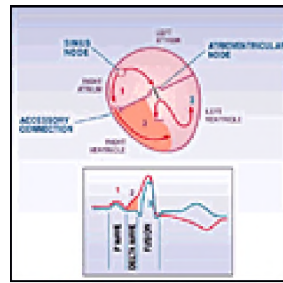
➤ Short PR: <0.12s

● Preexcitation syndromes:

■ WPW (Wolff-Parkinson-White) Syndrome: An accessory pathway



(called the "Kent" bundle) connects the right atrium to the right ventricle (see diagram below) or the left atrium to the left ventricle, and this permits early activation of the ventricles (*delta* wave) and a short PR interval.



[Click to view](#)

■ LGL (Lown-Ganong-Levine): An AV nodal bypass track into the His bundle exists, and this permits early activation of the ventricles without a *delta*-wave because the ventricular activation sequence is normal.

● AV Junctional Rhythms with retrograde atrial activation (inverted P waves in II, III, aVF): Retrograde P waves may occur *before* the QRS complex (usually with a short PR interval), *in* the QRS complex (i.e., hidden from view), or *after* the QRS complex (i.e., in the ST segment).

● Ectopic atrial rhythms originating near the AV node (the PR interval is short because atrial activation originates close to the AV node; the P wave morphology is different from the sinus P)

● Normal variant

➤ Prolonged PR: >0.20s

● First degree AV block (PR interval usually constant)

■ Intra-atrial conduction delay (uncommon)

■ Slowed conduction in AV node (most common site)

■ Slowed conduction in His bundle (rare)

■ Slowed conduction in bundle branch (when contralateral bundle is blocked)

● Second degree AV block (PR interval may be normal or prolonged; some P waves do not conduct)

■ Type I (Wenckebach): Increasing PR until nonconducted P wave occurs

■ Type II (Mobitz): Fixed PR intervals plus nonconducted P waves

● AV dissociation: Some PR's may appear prolonged, but the P waves and QRS complexes are dissociated (i.e., not married, but strangers passing in the night).

### 3. QRS Duration

(duration of QRS complex in frontal plane):

➤ Normal: 0.06 - 0.10s

➤ Prolonged QRS Duration (>0.10s):

● QRS duration 0.10 - 0.12s

■ Incomplete right or left bundle branch block

■ Nonspecific intraventricular conduction delay (IVCD)

■ Some cases of left anterior or posterior fascicular block

● QRS duration  $\geq 0.12s$

■ Complete RBBB or LBBB

■ Nonspecific IVCD

■ Ectopic rhythms originating in the ventricles (e.g., ventricular tachycardia, pacemaker rhythm)

## 4. QT Interval

(measured from beginning of QRS to end of T wave in the frontal plane)

➤ Normal: heart rate dependent (corrected QT =  $QT_c = \text{measured QT} \div \text{sq-root RR in seconds}$ ; upper limit for  $QT_c = 0.44 \text{ sec}$ )

➤ Long QT Syndrome - "LQTS" (based on upper limits for heart rate;  $QT_c \geq 0.47 \text{ sec}$  for males and  $\geq 0.48 \text{ sec}$  in females is diagnostic for *hereditary* LQTS in absence of other causes of increased QT)

● This abnormality may have important clinical implications since it usually indicates a state of increased vulnerability to malignant ventricular arrhythmias, syncope, and sudden death. The prototype arrhythmia of the *Long QT Interval Syndromes (LQTS)* is Torsade-de-pointes, a polymorphic ventricular tachycardia characterized by varying QRS morphology and amplitude around the isoelectric baseline. Causes of LQTS include the following:

- Drugs (many antiarrhythmics, tricyclics, phenothiazines, and others)
- Electrolyte abnormalities ( $\downarrow K^+$ ,  $\downarrow Ca^{++}$ ,  $\downarrow Mg^{++}$ )
- CNS disease (especially subarachnoid hemorrhage, stroke, trauma)
- Hereditary LQTS (e.g., Romano-Ward Syndrome)
- Coronary Heart Disease (some post-MI patients)

## 5. Frontal Plane QRS Axis

➤ Click here for brief tutorial in [Measuring QRS Axis](#)

➤ Normal: -30 degrees to +90 degrees

➤ Abnormalities in the QRS Axis:

● Left Axis Deviation (LAD):  $\geq -30^\circ$  (i.e., lead II is mostly 'negative')

■ Left Anterior Fascicular Block (LAFB): rS complex in leads II, III, aVF, small q in leads I and/or aVL, and axis  $-45^\circ$  to  $-90^\circ$

■ Some cases of inferior MI with Qr complex in lead II (making lead II 'negative')

■ Inferior MI + LAFB in same patient (QS or qrS complex in lead II)

■ Some cases of LVH

- Some cases of LBBB

- Ostium primum ASD and other endocardial cushion defects

- Some cases of WPW syndrome (large negative *delta* wave in lead II)

● Right Axis Deviation (RAD):  $\geq +90^\circ$  (i.e., lead I is mostly 'negative')

- Left Posterior Fascicular Block (LPFB): rS complex in lead I, qR in leads II, III, aVF (*however, must first exclude, on clinical basis, causes of right heart overload; these will also give same ECG picture of LPFB*)

- Many causes of right heart overload and pulmonary hypertension

- High lateral wall MI with Qr or QS complex in leads I and aVL

- Some cases of RBBB

- Some cases of WPW syndrome

- Children, teenagers, and some young adults

● Bizarre QRS axis:  $+150^\circ$  to  $-90^\circ$  (i.e., lead I and lead II are both negative)

- Consider limb lead error (usually right and left arm reversal)

- Dextrocardia

- Some cases of complex congenital heart disease (e.g., transposition)

- Some cases of ventricular tachycardia



Test your knowledge on lessons III and IV by [clicking here](#) (Internet connection required)



## V. ECG Rhythm Abnormalities

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I II III IV **V** VI VII VIII IX X XI XII

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### Topics for Study:

1. [Introduction to rhythm analysis](#)
2. [Supraventricular arrhythmias](#)
  - [Premature atrial complexes](#)
  - [Premature junctional complexes](#)
  - [Atrial fibrillation](#)
  - [Atrial flutter](#)
  - [Ectopic atrial tachycardia and rhythm](#)
  - [Multifocal atrial tachycardia](#)
  - [Paroxysmal supraventricular tachycardia](#)
  - [Junctional rhythms and tachycardias](#)
3. [Ventricular arrhythmias](#)
  - [Premature ventricular complexes \(PVCs\)](#)
  - [Aberrancy vs. ventricular ectopy](#)
  - [Ventricular tachycardia](#)
  - [Differential diagnosis of wide QRS tachycardias](#)
  - [Accelerated ventricular rhythms](#)
  - [Idioventricular rhythm](#)
  - [Ventricular parasystole](#)



[Test your knowledge on lesson V by clicking here](#) (Internet connection required)



## VI. ECG Conduction Abnormalities

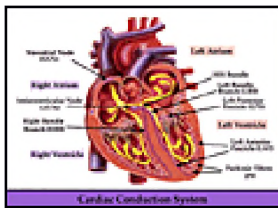
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I II III IV V VI VII VIII IX X XI XII

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### Topics for Study

1. [Introduction](#)
2. [Sino-Atrial Exit Block](#)
3. [Atrio-Ventricular \(AV\) Block](#)
  - [1st Degree AV Block](#)
  - [Type I \(Wenckebach\) 2nd Degree AV Block](#)
  - [Type II \(Mobitz\) 2nd Degree AV Block](#)
  - [Complete \(3rd Degree\) AV Block](#)
  - [AV Dissociation](#)
4. [Intraventricular Blocks](#)
  - [Right Bundle Branch Block](#)
  - [Left Bundle Branch Block](#)
  - [Left Anterior Fascicular Block](#)
  - [Left Posterior Fascicular Block](#)
  - [Bifascicular Blocks](#)
  - [Nonspecific Intraventricular Block](#)
  - [Wolff-Parkinson-White Preexcitation](#)



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## 1. Introduction:

This section considers all the important disorders of impulse conduction that may occur within the cardiac conduction system illustrated in the above diagram. Heart block can occur anywhere in the specialized conduction system beginning with the sino-atrial connections, the AV junction, the bundle branches and their fascicles, and ending in the distal ventricular Purkinje fibers. Disorders of conduction may manifest as slowed conduction (**1<sup>st</sup> degree**), intermittent conduction failure (**2<sup>nd</sup> degree**), or complete conduction failure (**3<sup>rd</sup> degree**). In addition, 2<sup>nd</sup> degree heart block occurs in two varieties: Type I (**Wenckebach**) and Type II (**Mobitz**). In Type I block there is **decremental conduction** which means that conduction velocity progressively slows down until failure of conduction occurs. Type II block is all or none. The term **exit block** is used to identify conduction delay or failure immediately distal to a pacemaker site. Sino-atrial (SA) block is an exit block. This section considers conduction disorders in the anatomical sequence that defines the cardiac conduction system; **so lets begin . . .**

## 2. Sino-Atrial Exit Block (SA Block):

➤ 2<sup>nd</sup> Degree SA Block: this is the only degree of SA block that can be recognized on the surface ECG (i.e., intermittent conduction failure between the sinus node and the right atrium). There are two types, although because of sinus arrhythmia they may be hard to differentiate. Furthermore, the differentiation is electrocardiographically interesting but not clinically important.

● Type I (SA Wenckebach): the following 3 rules represent the classic rules of Wenckebach, which were originally described for **Type I AV block**. The rules are the result of decremental conduction where the **increment** in conduction delay for each subsequent impulse gets smaller until conduction failure finally occurs. This declining increment results in the following findings:

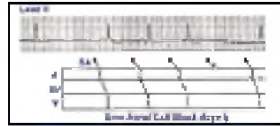
■ PP intervals gradually shorten until a pause occurs (i.e., the blocked sinus impulse fails to reach the atria)

■ The pause duration is **less than** the two preceding PP intervals



The PP interval following the pause is **greater than** the PP interval just before the pause

**Differential Diagnosis:** sinus arrhythmia without SA block. The following rhythm strip illustrates SA Wenckebach with a ladder diagram to show the progressive conduction delay between SA node and the atria. Note the similarity of this rhythm to marked sinus arrhythmia. (Remember, we cannot see SA events on the ECG, only the atrial response or P waves.)



[click here to view](#)

### Type II SA Block:

PP intervals fairly constant (unless sinus arrhythmia present) until conduction failure occurs.

The pause is approximately **twice** the basic PP interval



[Image not available](#)

## 3. Atrio-Ventricular (AV) Block

### ➤ Possible sites of AV block:

- AV node (most common)
- His bundle (uncommon)
- Bundle branch and fascicular divisions (in presence of already existing complete bundle branch block)

### ➤ 1<sup>st</sup> Degree AV Block: PR interval >0.20 sec; all P waves conduct to the ventricles.



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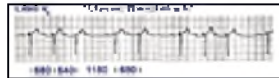
Wenckebach) and Type II AV block.



[click here to view](#)

In "classic" Type I (**Wenckebach**) AV block the PR interval gets longer (**by shorter increments**) until a nonconducted P wave occurs. The RR interval of the pause is **less than** the two preceding RR intervals, and the RR interval after the pause is **greater than** the RR interval before the pause. These are the **classic rules of Wenckebach** (atypical forms can occur). In Type II (**Mobitz**) AV block the PR intervals are constant until a nonconducted P wave occurs. There must be **two consecutive constant** PR intervals to diagnose Type II AV block (i.e., if there is 2:1 AV block we can't be sure if its type I or II). The RR interval of the pause is **equal to** the two preceding RR intervals.

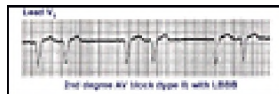
● Type I (Wenckebach) AV block (note the RR intervals in ms duration):



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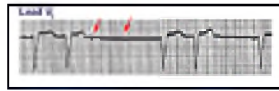
Type I AV block is **almost always** located in the AV node, which means that the QRS duration is usually narrow, unless there is preexisting bundle branch disease.

● Type II (Mobitz) AV block (note there are two consecutive constant PR intervals before the blocked P wave):



[click here to view](#)

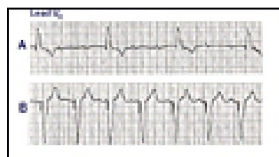
Type II AV block is ***almost always*** located in the bundle branches, which means that the QRS duration is wide indicating complete block of one bundle; the nonconducted P wave is blocked in the other bundle. In Type II block several consecutive P waves may be blocked as illustrated below:



[click here to view](#)

### ➤ Complete (3<sup>rd</sup> Degree) AV Block

- Usually see ***complete AV dissociation*** because the atria and ventricles are each controlled by separate pacemakers.
- Narrow QRS rhythm suggests a junctional escape focus for the ventricles with block above the pacemaker focus, usually in the AV node.
- Wide QRS rhythm suggests a ventricular escape focus (i.e., ***idioventricular rhythm***). This is seen in ECG 'A' below; ECG 'B' shows the treatment for 3<sup>rd</sup> degree AV block; i.e., a ***ventricular pacemaker***. The location of the block may be in the AV junction or bilaterally in the bundle branches.

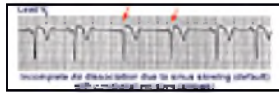


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### ➤ AV Dissociation (independent rhythms in atria and ventricles):

- Not synonymous with 3<sup>rd</sup> degree AV block, although AV block is one of the causes.
- May be ***complete*** or ***incomplete***. In complete AV dissociation the atria and ventricles are always independent of each other. In incomplete AV dissociation there is either intermittent atrial *capture* from the ventricular focus or ventricular *capture* from the atrial focus.
- There are ***three categories*** of AV dissociation (categories 1 & 2 are always ***incomplete*** AV dissociation):

1. Slowing of the primary pacemaker (i.e., SA node); subsidiary **escape** pacemaker takes over by **default**:



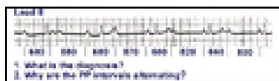
[click here to view](#)

2. Acceleration of a subsidiary pacemaker faster than sinus rhythm; takeover by **usurpation**:



[click here to view](#)

3. 2<sup>nd</sup> or 3<sup>rd</sup> degree AV block with escape rhythm from junctional focus or ventricular focus:



[click here to view](#)

In the above example of AV dissociation (3<sup>rd</sup> degree AV block with a junctional escape pacemaker) the PP intervals are alternating because of **ventriculophasic sinus arrhythmia** (phasic variation of vagal tone in the sinus node depending on the timing of ventricular contractions and blood flow near the carotid sinus).

## 4. Intraventricular Blocks

## ➤ Right Bundle Branch Block (RBBB):

- "Complete" RBBB has a QRS duration  $\geq 0.12$ s
- Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2<sup>nd</sup> half of QRS) are oriented rightward and anteriorly because the right ventricle is depolarized **after** the left ventricle. This means the following:

- Terminal R' wave in lead V1 (usually see rSR' complex) indicating late **anterior** forces

- Terminal S waves in leads I, aVL, V6 indicating late **rightward** forces

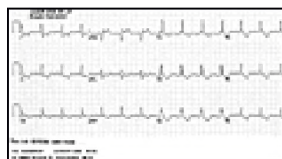
- Terminal R wave in lead aVR indicating late **rightward** forces

- The frontal plane QRS axis in RBBB should be in the normal range (i.e., -30 to +90 degrees). If left axis deviation is present, think about [left anterior fascicular block](#), and if right axis deviation is present, think about [left posterior fascicular block](#) in addition to the RBBB.

- "Incomplete" RBBB has a QRS duration of 0.10 - 0.12s with the same terminal QRS features. This is often a normal variant.

- The "normal" ST-T waves in RBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be negative or downwards; in leads with terminal S forces the ST-T should be positive or upwards. If the ST-T waves are in the **same direction** as the terminal QRS forces, they should be labeled **primary ST-T wave abnormalities**.

The ECG below illustrates primary ST-T wave abnormalities (leads I, II, aVR, V5, V6) in a patient with RBBB. ST-T wave abnormalities such as these may be related to ischemia, infarction, electrolyte abnormalities, medications, CNS disease, etc. (i.e., they are **nonspecific** and must be correlated with the patient's clinical status).



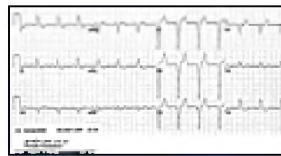
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## ➤ Left Bundle Branch Block (LBBB)

- "Complete" LBBB has a QRS duration  $\geq 0.12$ s
- Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2<sup>nd</sup> half of QRS) are oriented leftward and posteriorly because the left ventricle is depolarized **after** the right ventricle.

■ Terminal S waves in lead V1 indicating late **posterior** forces

■ Terminal R waves in lead I, aVL, V6 indicating late **leftward** forces; usually broad, monophasic R waves are seen in these leads as illustrated in the ECG below; in addition, poor R progression from V1 to V3 is common.



[click here to view](#)

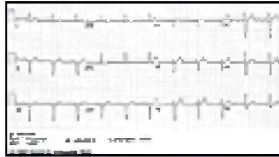
- The "normal" ST-T waves in LBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be downwards; in leads with terminal S forces the ST-T should be upwards. If the ST-T waves are in the **same direction** as the terminal QRS forces, they should be labeled **primary ST-T wave abnormalities**. In the above ECG the ST-T waves are "normal" for LBBB; i.e., they are **secondary** to the change in the ventricular depolarization sequence.

- "Incomplete" LBBB looks like LBBB but QRS duration = 0.10 to 0.12s, with less ST-T change. This is often a progression of LVH.

### ➤ Left Anterior Fascicular Block(LAFB)... the most common intraventricular conduction defect

- Left axis deviation in frontal plane, usually -45 to -90 degrees
- rS complexes in leads II, III, aVF
- Small q-wave in leads I **and/or** aVL
- R-peak time in lead aVL >0.04s, often with slurred R wave downstroke
- QRS duration usually <0.12s unless coexisting RBBB

- Usually see poor R progression in leads V1-V3 and deeper S waves in leads V5 and V6
- May **mimic** LVH voltage in lead aVL, and **mask** LVH voltage in leads V5 and V6.



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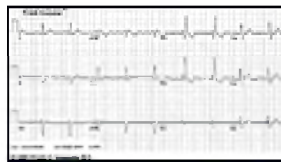
In this ECG, note -75 degree QRS axis, rS complexes in II, III, aVF, tiny q-wave in aVL, poor R progression V1-3, and late S waves in leads V5-6. QRS duration is normal, and there is a slight slur to the R wave downstroke in lead aVL.

#### ➤ Left Posterior Fascicular Block(LPFB).... Very rare intraventricular defect!

- Right axis deviation in the frontal plane (usually  $> +100$  degrees)
- rS complex in lead I
- qR complexes in leads II, III, aVF, with R in lead III  $>$  R in lead II
- QRS duration usually  $< 0.12$ s unless coexisting RBBB
- ***Must first exclude (on clinical grounds) other causes of right axis deviation such as cor pulmonale, pulmonary heart disease, pulmonary hypertension, etc., because these conditions can result in the identical ECG picture!***

#### ➤ Bifascicular Blocks

- RBBB plus either LAFB (common) **or** LPFB (uncommon)
- Features of RBBB plus frontal plane features of the fascicular block (axis deviation, etc.)



[click here to view](#)

The above ECG shows classic RBBB (note rSR' in V1) *plus* LAFB (note QRS axis = -45 degrees, rS in II, III, aVF; and small q in aVL).

### ➤ Nonspecific Intraventricular Conduction Defects (IVCD)

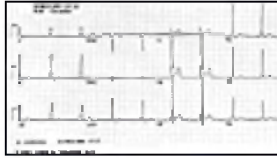
- QRS duration >0.10s indicating slowed conduction in the ventricles
- Criteria for specific bundle branch or fascicular blocks not met
- Causes of nonspecific IVCD's include:
  - Ventricular hypertrophy (especially LVH)
  - Myocardial infarction (so called *periinfarction blocks*)
  - Drugs, especially class IA and IC antiarrhythmics (e.g., quinidine, flecainide)
  - Hyperkalemia

### ➤ Wolff-Parkinson-White Preexcitation

- Although not a true IVCD, this condition causes widening of QRS complex and, therefore, deserves to be considered here
- QRS complex represents a *fusion* between *two* ventricular activation fronts:
  - Early ventricular activation in region of the accessory AV pathway (*Bundle of Kent*)
  - Ventricular activation through the normal AV junction, bundle branch system
  - ECG criteria include all of the following:
    - Short PR interval (<0.12s)



- Initial slurring of QRS complex (**delta wave**) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway
- Prolonged QRS duration (usually >0.10s)
- Secondary ST-T changes due to the altered ventricular activation sequence



[click here to view](#)

● QRS morphology, including polarity of delta wave depends on the particular location of the accessory pathway as well as on the relative proportion of the QRS complex that is due to early ventricular activation (i.e., degree of fusion).

● Delta waves, if negative in polarity, may mimic infarct Q waves and result in false positive diagnosis of myocardial infarction.



[Test your knowledge on lesson VI by clicking here](#) (Requires and Internet connection)



## VII. Atrial Enlargement

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I II III IV V VI VII VIII IX X XI XII

Topics for study:

1. [Right Atrial Enlargement \(RAE\)](#)
2. [Left Atrial Enlargement \(LAE\)](#)
3. [Bi-Atrial Enlargement \(BAE\)](#)

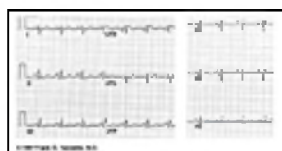
### 1. Right Atrial Enlargement (RAE)

➤ P wave amplitude >2.5 mm in II and/or >1.5 mm in V1 (these criteria are not very specific or sensitive)

➤ Better criteria can be derived from the QRS complex; these QRS changes are due to both the high incidence of RVH when RAE is present, and the RV displacement by an enlarged right atrium.

● QR, Qr, qR, or qRs morphology in lead V1 (in absence of coronary heart disease)

● QRS voltage in V1 is <5 mm and V2/V1 voltage ratio is >6 (Sensitivity = 50%; Specificity = 90%)



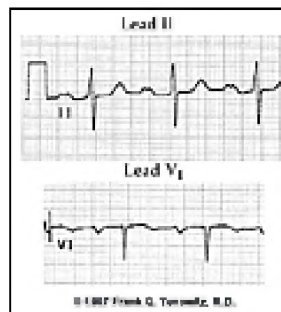
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In the above ECG, note the tall P waves in Lead II, and the Qr

wave in Lead V1.

## 2. Left Atrial Enlargement (LAE)

- P wave duration  $\geq 0.12$ s in frontal plane (usually lead II)
- Notched P wave in limb leads with the inter-peak duration  $\geq 0.04$ s
- Terminal P negativity in lead V1 (i.e., "P-terminal force") duration  $\geq 0.04$ s, depth  $\geq 1$  mm.
- Sensitivity = 50%; Specificity = 90%



[click here to view](#)

## 3. Bi-Atrial Enlargement (BAE)

- Features of both RAE and LAE in same ECG
- P wave in lead II  $> 2.5$  mm tall and  $> 0.12$ s in duration
- Initial positive component of P wave in V1  $> 1.5$  mm tall and prominent P-terminal force





## VIII. Ventricular Hypertrophy

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I II III IV V VI VII VIII IX X XI XII

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Topics for study:

1. [Introduction](#)
2. [Left Ventricular Hypertrophy \(LVH\)](#)
3. [Right Ventricular Hypertrophy \(RVH\)](#)
4. [Biventricular Hypertrophy](#)

### 1. Introductory Information:

➤ The ECG criteria for diagnosing right or left ventricular hypertrophy are very insensitive(i.e., sensitivity ~50%, which means that ~50% of patients with ventricular hypertrophy cannot be recognized by ECG criteria). However, the criteria are very specific(i.e., specificity >90%, which means if the criteria are met, it is very likely that ventricular hypertrophy is present).

### 2. Left Ventricular Hypertrophy (LVH)

➤ GeneralECG features include:

●  $\geq$  QRS amplitude (voltage criteria; i.e., tall R-waves in LV leads, deep S-waves in RV leads)

- Delayed intrinsicoid deflection in V6 (i.e., time from QRS onset to peak R is  $\geq 0.05$  sec)
- Widened QRS/T angle (i.e., *left ventricular strain pattern*, or ST-T oriented opposite to QRS direction)
- Leftward shift in frontal plane QRS axis
- Evidence for [left atrial enlargement \(LAE\) \(lessonVII\)](#)

➤ ESTES Criteria for LVH ("diagnostic",  $\geq 5$  points; "probable", 4 points)

+ECG Criteria	Points
<b>Voltage Criteria (any of):</b> a. R or S in limb leads $\geq 20$ mm b. S in V1 or V2 $\geq 30$ mm c. R in V5 or V6 $\geq 30$ mm	3 points
<b>ST-T Abnormalities:</b> Without digitalis	3 points
With digitalis	1 point
Left Atrial Enlargement in V1	3 points
Left axis deviation	2 points
QRS duration 0.09 sec	1 point
Delayed intrinsicoid deflection in V5 or V6 ( $>0.05$ sec)	1 point

➤ CORNELL Voltage Criteria for LVH (sensitivity = 22%, specificity = 95%)

- S in V3 + R in aVL  $> 24$  mm (men)
- S in V3 + R in aVL  $> 20$  mm (women)

➤ Other Voltage Criteria for LVH

- Limb-lead voltage criteria:
  - R in aVL  $\geq 11$  mm or, if left axis deviation, R in aVL  $\geq 13$  mm plus S in III  $\geq 15$  mm
  - R in I + S in III  $> 25$  mm

- Chest-lead voltage criteria:

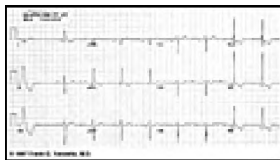
■ S in V1 + R in V5 or V6  $\geq$  35 mm

● Example 1: (Limb-lead Voltage Criteria; e.g., R in aVL >11 mm; note wide QRS/T angle)



[click here to view](#)

● Example 2: (ESTES Criteria: 3 points for voltage in V5, 3 points for ST-T changes)



[click here to view](#)

(Note also the left axis deviation of -40 degrees, and left atrial enlargement)

### 3. Right Ventricular Hypertrophy

➤ GeneralECG features include:

- Right axis deviation (>90 degrees)
- Tall R-waves in RV leads; deep S-waves in LV leads
- Slight increase in QRS duration
- ST-T changes directed opposite to QRS direction (i.e., wide QRS/T angle)
- May see incomplete RBBB pattern or qR pattern in V1
- Evidence of [right atrial enlargement \(RAE\) \(lessonVII\)](#)

➤ Specific ECG features (assumes normal calibration of 1 mV = 10 mm):

● Any one or more of the following (if QRS duration < 0.12 sec):

■ Right axis deviation (>90 degrees) in presence of disease capable of causing RVH

■ R in aVR  $\geq$  5 mm, or

■ R in aVR > Q in aVR

● Any one of the following in lead V1:

■ R/S ratio > 1 and negative T wave

■ qR pattern

■ R > 6 mm, or S < 2mm, or rSR' with R' > 10 mm

● Other chest lead criteria:

■ R in V1 + S in V5 (or V6) 10 mm

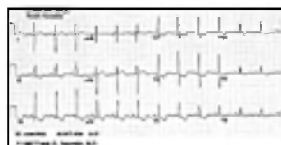
■ R/S ratio in V5 or V6 < 1

■ R in V5 or V6 < 5 mm

■ S in V5 or V6 > 7 mm

● ST segment depression and T wave inversion in right precordial leads is usually seen in severe RVH such as in pulmonary stenosis and pulmonary hypertension.

➤ Example #1: (note RAD +105 degrees; RAE; R in V1 > 6 mm; R in aVR > 5 mm)



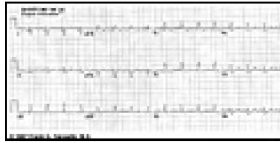
[click here to view](#)

➤ Example #2:(more subtle RVH: note RAD +100 degrees; RAE; Qr complex in V1 rather than qR is atypical)



[click here to view](#)

➤ Example #3:(note: RAD +120 degrees, qR in V1; R/S ratio in V6 <1)



[click here to view](#)

#### 4. Biventricular Hypertrophy (difficult ECG diagnosis to make)

➤ In the presence of LAE any one of the following suggests this diagnosis:

- R/S ratio in V5 or V6 < 1
- S in V5 or V6 > 6 mm
- RAD (>90 degrees)

➤ Other suggestive ECG findings:

- Criteria for LVH and RVH both met
- LVH criteria met and RAD or RAE present



[Test your knowledge on lessons VII and VIII by clicking here](#) (Requires Internet)





## IX. Myocardial Infarction

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I II III IV V VI VII VIII IX X XI XII

### Topics for study:

1. [Introduction](#) (Read this first)
2. [Inferior Q-Wave MI Family](#)
3. [Anterior Q-Wave MI Family](#)
4. [MI + Bundle Branch Block](#)
5. [Non Q-Wave MI](#)
6. [The Pseudoinfarctions](#)
7. [Miscellaneous QRS Abnormalities](#)

### 1. Introduction to ECG Recognition of Myocardial Infarction

➤ When myocardial blood supply is abruptly reduced or cut off to a region of the heart, a sequence of injurious events occur beginning with subendocardial or transmural ischemia, followed by necrosis, and eventual fibrosis (scarring) if the blood supply isn't restored in an appropriate period of time. Rupture of an atherosclerotic plaque followed by acute coronary thrombosis is the usual mechanism of acute MI. The ECG changes reflecting this sequence usually follow a well-known pattern depending on the location and size of the MI. MI's resulting from total coronary occlusion result in more homogeneous tissue damage and are usually reflected by a **Q-wave MI pattern** on the ECG. MI's resulting from subtotal occlusion result in more heterogeneous damage, which may be evidenced by a [non Q-wave MI](#) pattern on the ECG. **Two-thirds of MI's presenting to emergency rooms evolve to non-Q wave MI's, most having ST segment depression or T wave inversion.**

➤ Most MI's are located in the **left ventricle**. In the setting of a proximal right coronary artery occlusion, however, up to 50% may also have a component of [right ventricular infarction](#) as well. Right-sided chest leads are necessary to recognize RV MI.

➤ In general, the more leads of the 12-lead ECG with MI changes (Q waves and ST

elevation), the larger the infarct size and the worse the prognosis. Additional leads on the back, V7-9 (horizontal to V6), may be used to improve the recognition of true posterior MI.

➤ The left anterior descending coronary artery (LAD) and its branches usually supply the anterior and anterolateral walls of the left ventricle and the anterior two-thirds of the septum. The left circumflex coronary artery (LCX) and its branches usually supply the posterolateral wall of the left ventricle. The right coronary artery (RCA) supplies the right ventricle, the inferior (diaphragmatic) and true posterior walls of the left ventricle, and the posterior third of the septum. The RCA also gives off the AV nodal coronary artery in 85-90% of individuals; in the remaining 10-15%, this artery is a branch of the LCX.

➤ Usual ECG evolution of a Q-wave MI; not all of the following patterns may be seen; the time from onset of MI to the final pattern is quite variable and related to the size of MI, the rapidity of reperfusion (if any), and the location of the MI.

● A. Normal ECG prior to MI

● B. Hyperacute T wave changes - increased T wave amplitude and width; may also see ST elevation

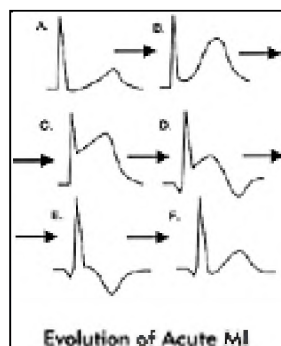
● C. Marked ST elevation with hyperacute T wave changes (transmural injury)

● D. Pathologic Q waves, less ST elevation, terminal T wave inversion (necrosis)

■ (Pathologic Q waves are usually defined as duration  $\geq 0.04$  s or  $>25\%$  of R-wave amplitude)

● E. Pathologic Q waves, T wave inversion (necrosis and fibrosis)

● F. Pathologic Q waves, upright T waves (fibrosis)



[click here to view](#)

## 2. Inferior MI Family of Q-wave MI's

(includes inferior, true posterior, and right ventricular MI's)

## ➤ Inferior MI

- Pathologic Q waves and evolving ST-T changes in leads II, III, aVF
- Q waves usually largest in lead III, next largest in lead aVF, and smallest in lead II
- Example #1: frontal plane leads with fully evolved inferior MI (note Q-waves, residual ST elevation, and T inversion in II, III, aVF)



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- Example #2: Old inferior MI (note largest Q in lead III, next largest in aVF, and smallest in lead II)



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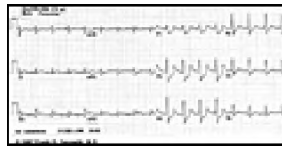
## ➤ True posterior MI

- ECG changes are seen in anterior precordial leads V1-3, but are the mirror image of an anteroseptal MI:

- Increased R wave amplitude and duration (i.e., a "pathologic R wave" is a mirror image of a pathologic Q)
- R/S ratio in V1 or V2 >1 (i.e., prominent anterior forces)
- Hyperacute ST-T wave changes: i.e., ST depression and large, inverted T waves in V1-3
- Late normalization of ST-T with symmetrical upright T waves in V1-3

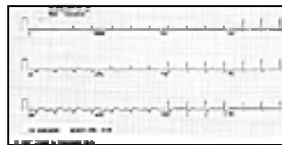
- Often seen with inferior MI (i.e., "inferoposterior MI")

- **Example #1:** Acute inferoposterior MI (note tall R waves V1-3, marked ST depression V1-3, ST elevation in II, III, aVF)



[click here to view](#)

- **Example #2:** Old inferoposterior MI (note tall R in V1-3, upright T waves and inferior Q waves)



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- **Example #3:** Old posterolateral MI (precordial leads): note tall R waves and upright T's in V1-3, and loss of R in V6

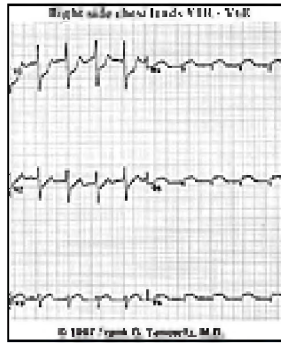


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➤ Right Ventricular MI (only seen with proximal right coronary occlusion; i.e., with inferior family MI's)

- ECG findings usually require additional leads on right chest (V1R to V6R, analogous to the left chest leads)

- ST elevation,  $\geq 1$ mm, in right chest leads, especially V4R (see below)

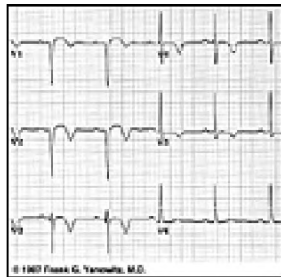


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### 3. Anterior Family of Q-wave MI's

#### ➤ Anteroseptal MI

- Q, QS, or qrS complexes in leads V1-V3 (V4)
- Evolving ST-T changes
- Example: Fully evolved anteroseptal MI (note QS waves in V1-2, qrS complex in V3, plus ST-T wave changes)



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#### ➤ Anterior MI (similar changes, but usually V1 is spared; if V4-6 involved call it "anterolateral")

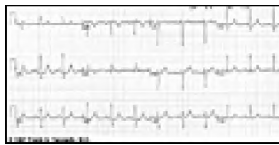
- Example: Acute anterior or anterolateral MI (note Q's V2-6 plus hyperacute ST-T changes)



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➤ High Lateral MI (typical MI features seen in leads I and/or aVL)

● Example: note Q-wave, slight ST elevation, and T inversion in lead aVL



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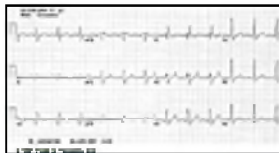
(Note also the slight U-wave inversion in leads II, III, aVF, V4-6, a strong marker for coronary disease)

## 4. MI with Bundle Branch Block

➤ MI + Right Bundle Branch Block

● Usually easy to recognize because Q waves and ST-T changes are not altered by the RBBB

● Example #1: Inferior MI + RBBB (note Q's in II, III, aVF and rSR' in lead V1)



[click here to view](#)

● Example #2: Anteroseptal MI with RBBB (note Q's in leads V1-V3, terminal R wave in V1, fat S wave in V6)



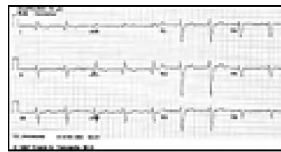
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## ➤ MI + Left Bundle Branch Block

● Often a difficult ECG diagnosis because in LBBB the right ventricle is activated first and left ventricular infarct Q waves may not appear at the beginning of the QRS complex (unless the septum is involved).

● Suggested ECG features, not all of which are specific for MI include:

■ Q waves of any size in two or more of leads I, aVL, V5, or V6 (See below: one of the most reliable signs and probably indicates *septal* infarction, because the septum is activated early from the right ventricular side in LBBB)



[click here to view](#)

■ Reversal of the usual R wave progression in precordial leads (see above )

■ Notching of the downstroke of the S wave in precordial leads to the right of the transition zone (i.e., before QRS changes from a predominate S wave complex to a predominate R wave complex); this may be a Q-wave equivalent.

■ Notching of the upstroke of the S wave in precordial leads to the right of the transition zone (another Q-wave equivalent).

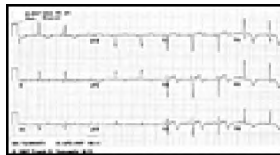
■ rSR' complex in leads I, V5 or V6 (the S is a Q-wave equivalent occurring in the middle of the QRS complex)

■ RS complex in V5-6 rather than the usual monophasic R waves seen in uncomplicated LBBB; (the S is a Q-wave equivalent).

■ "Primary" ST-T wave changes (i.e., ST-T changes in the same direction as the QRS complex rather than the usual "secondary" ST-T changes seen in uncomplicated LBBB); these changes may reflect an acute, evolving MI.

## 5. Non-Q Wave MI

- Recognized by evolving ST-T changes over time without the formation of pathologic Q waves (in a patient with typical chest pain symptoms and/or elevation in myocardial-specific enzymes)
- Although it is tempting to localize the non-Q MI by the particular leads showing ST-T changes, this is probably only valid for the ST segment elevation pattern
- Evolving ST-T changes may include any of the following patterns:
  - Convex downward ST segment depression only (common)
  - Convex upwards or straight ST segment elevation only (uncommon)
  - Symmetrical T wave inversion only (common)
  - Combinations of above changes
  - Example: Anterolateral ST-T wave changes



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## 6. The Pseudoinfarcts

- These are ECG conditions that mimic myocardial infarction either by simulating pathologic Q or QS waves or mimicking the typical ST-T changes of acute MI.
  - WPW preexcitation (*negative* delta wave may mimic pathologic Q waves)
  - IHSS (septal hypertrophy may make normal septal Q waves



"fatter" thereby mimicking pathologic Q waves)

- LVH (may have QS pattern or poor R wave progression in leads V1-3)

- RVH (tall R waves in V1 or V2 may mimic true posterior MI)

- Complete or incomplete LBBB (QS waves or poor R wave progression in leads V1-3)

- Pneumothorax (loss of right precordial R waves)

- Pulmonary emphysema and cor pulmonale (loss of R waves V1-3 and/or inferior Q waves with right axis deviation)

- Left anterior fascicular block (may see small q-waves in anterior chest leads)

- Acute pericarditis (the ST segment elevation may mimic acute transmural injury)

- Central nervous system disease (may mimic non-Q wave MI by causing diffuse ST-T wave changes)

## 7. Miscellaneous Abnormalities of the QRS Complex:

- The differential diagnosis of these QRS abnormalities depend on other ECG findings as well as clinical patient information

- Poor R Wave Progression - defined as loss of, or no R waves in leads V1-3 ( $R \leq 2\text{mm}$ ):

- Normal variant (if the rest of the ECG is normal)

- LVH (look for voltage criteria and ST-T changes of LV "strain")

- Complete or incomplete LBBB (increased QRS duration)

- Left anterior fascicular block (should see LAD in frontal plane)

- [Anterior or anteroseptal MI](#)

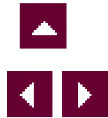
- Emphysema and COPD (look for R/S ratio in V5-6  $< 1$ )

- Diffuse infiltrative or myopathic processes

- WPW preexcitation (look for *delta waves*, short PR)

➤ Prominent Anterior Forces - defined as R/S ratio  $>1$  in V1 or V2

- Normal variant (if rest of the ECG is normal)
- [True posterior MI](#) (look for evidence of inferior MI)
- RVH (should see RAD in frontal plane and/or P-pulmonale)
- Complete or incomplete RBBB (look for rSR' in V1)
- WPW preexcitation (look for *delta waves*, short PR)



[Test your knowledge on lesson IX by clicking here](#) (Requires Internet)



## X. ST Segment Abnormalities

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I II III IV V VI VII VIII IX X XI XII

Topics for study:

1. [General Introduction to ST-T and U Wave Abnormalities](#)
2. [ST Segment Elevation](#)
3. [ST Segment Depression](#)

### 1. General Introduction to ST, T, and U wave abnormalities

➤ Basic Concept: the *specificity* of ST-T and U wave abnormalities is provided more by the clinical circumstances in which the ECG changes are found than by the particular changes themselves. Thus the term, *nonspecific ST-T wave abnormalities*, is frequently used when the clinical data are not available to correlate with the ECG findings. This does not mean that the ECG changes are unimportant! It is the responsibility of the clinician providing care for the patient to ascertain the importance of the ECG findings.

➤ Factors affecting the ST-T and U wave configuration include:

- Intrinsic myocardial disease (e.g., myocarditis, ischemia, infarction, infiltrative or myopathic processes)
- Drugs (e.g., digoxin, quinidine, tricyclics, and many others)
- Electrolyte abnormalities of potassium, magnesium, calcium
- Neurogenic factors (e.g., stroke, hemorrhage, trauma, tumor, etc.)
- Metabolic factors (e.g., hypoglycemia, hyperventilation)

- Atrial repolarization (e.g., at fast heart rates the atrial T wave may pull down the beginning of the ST segment)
- Ventricular conduction abnormalities and rhythms originating in the ventricles

➤ "Secondary" ST-T Wave changes (these are normal ST-T wave changes solely due to alterations in the sequence of ventricular activation)

- ST-T changes seen in bundle branch blocks (generally the ST-T polarity is opposite to the major or terminal deflection of the QRS)
- ST-T changes seen in fascicular block
- ST-T changes seen in nonspecific IVCD
- ST-T changes seen in WPW preexcitation
- ST-T changes in PVCs, ventricular arrhythmias, and ventricular paced beats

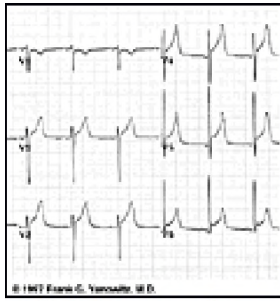
➤ "Primary" ST-T Wave Abnormalities (ST-T wave changes that are independent of changes in ventricular activation and that may be the result of global or segmental pathologic processes that affect ventricular repolarization)

- Drug effects (e.g., digoxin, quinidine, etc)
- Electrolyte abnormalities (e.g., hypokalemia)
- Ischemia, infarction, inflammation, etc
- Neurogenic effects (e.g., subarachnoid hemorrhage causing long QT)

## 2. Differential Diagnosis of ST Segment Elevation

➤ Normal Variant "Early Repolarization" (usually concave upwards, ending with symmetrical, large, upright T waves)

- Example #1: "Early Repolarization": note high take off of the ST segment in leads V4-6; the ST elevation in V2-3 is generally seen in most normal ECG's; the ST elevation in V2-6 is concave upwards, another characteristic of this normal variant.



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➤ Ischemic Heart Disease (usually convex upwards, or straightened)

- Acute transmural injury - as in this acute anterior MI



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- Persistent ST elevation after acute MI suggests ventricular aneurysm
- ST elevation may also be seen as a manifestation of Prinzmetal's (variant) angina (coronary artery spasm)
- ST elevation during exercise testing suggests extremely tight coronary artery stenosis or spasm (transmural ischemia)

➤ Acute Pericarditis

- Concave upwards ST elevation in most leads except aVR
- No reciprocal ST segment depression (except in aVR)
- Unlike "early repolarization", T waves are usually low amplitude, and heart rate is usually increased.
- May see PR segment depression, a manifestation of atrial injury

➤ Other Causes:

- Left ventricular hypertrophy (in right precordial leads with large S-waves)
- Left bundle branch block (in right precordial leads with large S-waves)
- Advanced hyperkalemia
- Hypothermia (prominent J-waves or Osborne waves)

### 3. Differential Diagnosis of ST Segment Depression

➤ Normal variants or artifacts:

- Pseudo-ST-depression (wandering baseline due to poor skin-electrode contact)
- Physiologic J-junctional depression with sinus tachycardia (most likely due to atrial repolarization)
- Hyperventilation-induced ST segment depression

➤ Ischemic heart disease

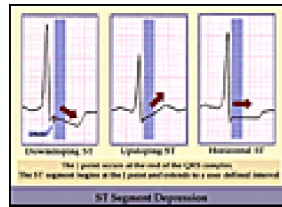
- Subendocardial ischemia (exercise induced or during angina attack - as illustrated below)



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Note: "horizontal" ST depression in lead V6

■ ST segment depression is often characterized as "horizontal", "upsloping", or "downsloping"



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Note: "Upsloping" ST depression is not an ischemic abnormality

- Non Q-wave MI
- Reciprocal changes in acute Q-wave MI (e.g., ST depression in leads I & aVL with acute inferior MI)

➤ Nonischemic causes of ST depression

- RVH (right precordial leads) or LVH (left precordial leads, I, aVL)
- Digoxin effect on ECG
- Hypokalemia
- Mitral valve prolapse (some cases)
- CNS disease
- Secondary ST segment changes with IV conduction abnormalities (e.g., RBBB, LBBB, WPW, etc)





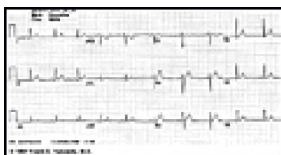
## XI. T Wave Abnormalities

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I II III IV V VI VII VIII IX X **XI** XII

### INTRODUCTION:

The T wave is the most labile wave in the ECG. T wave changes including low-amplitude T waves and abnormally inverted T waves may be the result of many cardiac and non-cardiac conditions. The normal T wave is usually in the same direction as the QRS except in the right precordial leads (see V2 below). Also, the normal T wave is *asymmetric* with the first half moving more slowly than the second half. In the normal ECG (see below) the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR. The other leads are variable depending on the direction of the QRS and the age of the patient.

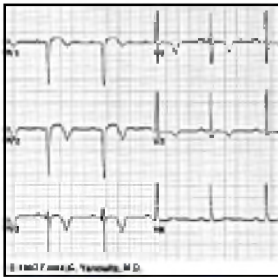


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### Differential Diagnosis of T Wave Inversion

➤ Q wave and non-Q wave MI (e.g., evolving anteroseptal MI):





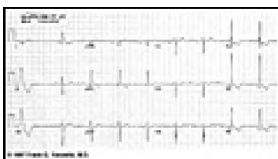
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- Myocardial ischemia
- Subacute or old pericarditis
- Myocarditis
- Myocardial contusion (from trauma)
- CNS disease causing long QT interval (especially subarachnoid hemorrhage; see below):



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- Idiopathic apical hypertrophy (a rare form of hypertrophic cardiomyopathy)
- Mitral valve prolapse
- Digoxin effect
- RVH and LVH with "strain" (see below: T wave inversion in leads aVL, V4-6 in LVH)



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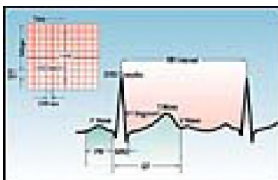
## XII. Nice Seeing "U" Again

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I II III IV V VI VII VIII IX X XI XII

### Introduction:

The U wave is the only remaining enigma of the ECG, and probably not for long. The origin of the U wave is still in question, although most authorities correlate the U wave with electrophysiologic events called "afterdepolarizations" in the ventricles. These afterdepolarizations can be the source of arrhythmias caused by "triggered automaticity" including *torsade de pointes*. The normal U wave has the same polarity as the T wave and is usually less than one-third the amplitude of the T wave. U waves are usually best seen in the right precordial leads especially V2 and V3. The normal U wave is asymmetric with the ascending limb moving more rapidly than the descending limb (just the opposite of the normal T wave).



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### Differential Diagnosis of U Wave Abnormalities

#### ➤ Prominent upright U waves

- Sinus bradycardia accentuates the U wave
- Hypokalemia (remember the triad of ST segment depression, low amplitude T

waves, and prominent U waves)

- Quinidine and other type 1A antiarrhythmics

- CNS disease with long QT intervals (often the T and U fuse to form a giant "T-U fusion wave")



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(E.g., lead II, III, V4-6)

- LVH (right precordial leads with deep S waves)

- Mitral valve prolapse (some cases)

- Hyperthyroidism

#### ➤ Negative or "inverted" U waves

- Ischemic heart disease (often indicating left main or LAD disease)

- Myocardial infarction (in leads with pathologic Q waves)

- During episode of acute ischemia (angina or exercise-induced ischemia)

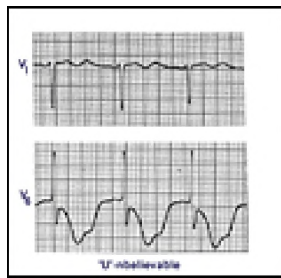
- Post extrasystolic in patients with coronary heart disease

- During coronary artery spasm (Prinzmetal's angina)

- Nonischemic causes

- Some cases of LVH or RVH (usually in leads with prominent R waves)

- Some patients with LQTS (see below: Lead V6 shows giant negative TU fusion wave in patient with LQTS; a prominent upright U wave is seen in Lead V1)



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## Lesson V (cont) Introduction to ECG Rhythm Analysis

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### Things to Consider When Analyzing Arrhythmias

Arrhythmias may be seen on 12-lead ECGs or on strips of one or more leads. Some arrhythmias are obvious at first glance and don't require intense analysis. Others, however, are more fun! They require detective work, i.e., logical thinking based on a knowledge of cardiac electrophysiology. The analysis should begin with identifying characteristics of **impulse formation** (if known) as well as **impulse conduction**. Here are some things to think about:

1. [Descriptors of impulse formation](#) (i.e. the pacemaker)
2. [Descriptors of impulse conduction](#) (i.e., how it moves through the heart)

### 1. Descriptors of **impulse formation**

(i.e. the pacemaker or region of impulse formation)

➤ Site of origin (i.e., where is the abnormal rhythm coming from?)

● Sinus Node (e.g., sinus tachycardia)

- Atria (e.g., PAC)
- AV junction (e.g., junctional escape rhythm)
- Ventricles (e.g., PVC)

➤ Rate (i.e., relative to the "expected rate" for that pacemaker location)

- Accelerated - faster than expected (e.g., accelerated junctional rhythm @ 75bpm)
- Slower than expected (e.g., marked sinus bradycardia @ 40bpm)
- Normal (e.g., junctional escape rhythm)

➤ Regularity of ventricular or atrial response

- Regular (e.g., PSVT)
- Regular irregularity (e.g., ventricular bigeminy)
- Irregular irregularity (e.g., atrial fibrillation or MAT)
- Irregular (e.g., multifocal PVCs)

➤ Onset (i.e., how does the arrhythmia begin?)

- Active onset (i.e., begins prematurely as with PAC or PVC)
- Passive onset (e.g., ventricular escape beat or rhythm)

## 2. Descriptors of *impulse conduction*

(i.e., how abnormal rhythm conducts through the heart)

- Antegrade (forward) vs. retrograde (backward) conduction
- Conduction delays or blocks: i.e., 1<sup>st</sup>, 2<sup>nd</sup> (type I or II), 3<sup>rd</sup> degree blocks

### ➤ Sites of potential conduction delay

- Sino-Atrial (SA) exit block (can only recognize 2<sup>nd</sup> degree SA block on ECG)
- Intra-atrial delay (usually not recognized)
- AV conduction delays (common)
- IV blocks (e.g., bundle branch or fascicular blocks)

Now let's explore some real rhythm abnormalities..... [\(Return to Lesson V\)](#)





## Lesson V (cont) Supraventricular Arrhythmias

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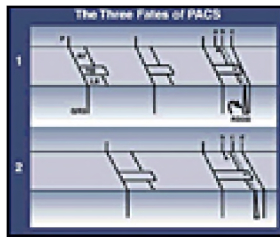
[Return to the beginning of Lesson V](#)

1. [Premature atrial complexes](#)
2. [Premature junctional complexes](#)
3. [Atrial fibrillation](#)
4. [Atrial flutter](#)
5. [Ectopic atrial tachycardia and rhythm](#)
6. [Multifocal atrial tachycardia](#)
7. [Paroxysmal supraventricular tachycardia](#)
8. [Junctional rhythms and tachycardias](#)

### 1. Premature atrial complexes

- Occur as single or repetitive events and have unifocal or multifocal origins.
- The ectopic P wave (called P') is often hidden in the ST-T wave of the preceding beat. (Dr. Marriott, master ECG teacher and author, likes to say: "***Cherchez le P on let T***" which in French means: "Search for the P on the T wave", but it's more sexy in French!)
- The P-R interval is normal or prolonged because the AV junction is often partially refractory when the premature impulse enters it.
- PAC's can have ***three different outcomes*** depending on the degree of prematurity (i.e., coupling interval from previous P wave), and the preceding cycle length. This is illustrated in the "ladder" diagram where normal sinus beats (P) are followed by three possible PACs; in the diagram the refractory periods of the AV node and bundle branches are indicated by the width of the boxes):





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A "ladder" diagram is an easy way of conceptualizing the conduction of impulses through the heart, and the resulting complexes (i.e., P waves and QRS waves).

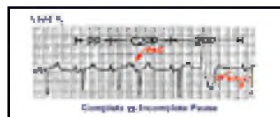
● Outcome #1. Nonconducted(blocked); i.e., no QRS complex because the PAC finds AV node still refractory. (see PAC labeled 'a' in the upper diagram 1)

● Outcome #2. Conducted with aberration; i.e., PAC makes it into the ventricles but finds one or more of the conducting fascicles or bundle branches refractory. The resulting QRS is usually wide, and is sometimes called an **Ashman beat**(see PAC 'b' in diagram 1)

● Outcome #3. Normal conduction; i.e., similar to other QRS complexes in the ECG. (See PAC 'c' in the diagram 1)

● In the diagram 2, seen above, the cycle length (i.e., PP interval) has increased (slower heart rate), and this results in increased refractoriness of all the structures in the conduction system (i.e., wider boxes). PAC 'b' now can't get through the AV node and is nonconducted; PAC 'c' is now blocked in the right bundle branch and results in a RBBB QRS complex (aberrant conduction); PAC 'd' is far enough away to conduct normally. **Therefore, the fate of a PAC depends on 1) the coupling interval from the last P wave and 2) the preceding cycle length or heart rate.**

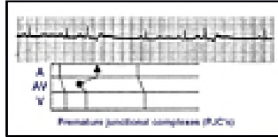
➤ The pause after a PAC is usually **incomplete**; i.e., the PAC usually enters the sinus node and resets its timing, causing the next sinus P to appear earlier than expected. (PVCs, on the other hand, are usually followed by a **complete** pause because the PVC does not usually perturb the sinus node; see ECG below.)



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## 2. Premature junctional complexes

- Similar to PAC's in clinical implications, but occur less frequently.
- The PJC focus, located in the AV junction, captures the atria (retrograde) and the ventricles (antegrade). The retrograde P wave may appear before, during, or after the QRS complex; if before, the PR interval is usually short (i.e., <0.12 s). The ECG tracing and ladder diagram shown below illustrates two classic PJC's with retrograde P waves following the QRS.



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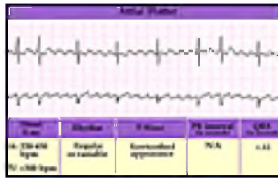
### 3. Atrial Fibrillation (A-fib)



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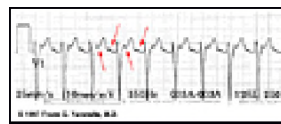
- Atrial activity is poorly defined; may see coarse or fine undulations *ono* atrial activity at all. If atrial activity is seen, it resembles an **old saw**(when compared to atrial flutter that often resembles a **new saw**).
- Ventricular response is **irregularly irregular** and may be **fast** (HR >100 bpm, indicates inadequate rate control), **moderate** (HR = 60-100 bpm), or **slow** (HR <60 bpm, indicates excessive rate control, AV node disease, or drug toxicity).
- A **regular** ventricular response with A-fib usually indicates complete AV block with an escape or accelerated ectopic pacemaker originating in the AV junction or ventricles (i.e., must consider digoxin toxicity or AV node disease).
- The differential diagnosis includes atrial flutter with an irregular ventricular response **and** multifocal atrial tachycardia (MAT), which is usually irregularly irregular. The differential diagnosis may be hard to make from a single lead rhythm strip; the 12-lead ECG is best for differentiating these three arrhythmias.

### 4. Atrial Flutter (A-flutter):



[click here to view](#)

- Regular atrial activity with a "clean" **saw-tooth** appearance in leads II, III, aVF, and usually discrete 'P' waves in lead V1. The atrial rate is usually about 300/min, but may be as slow as 150-200/min or as fast as 400-450/min.
- Untreated A-flutter often presents with a 2:1 A-V conduction ratio. This is the most commonly missed supraventricular tachycardia because the flutter waves are often difficult to find when there is 2:1 ratio. Therefore, always **think "atrial flutter with 2:1 block" whenever there is a regular supraventricular tachycardia @ ~150 bpm!** (You won't miss it if you look for it in a 12-lead ECG)



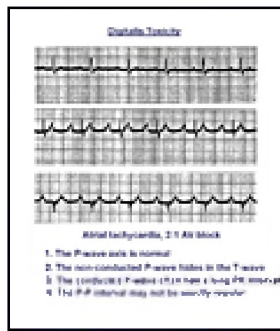
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In this ECG rhythm strip, arrows point to atrial flutter waves @ 280bpm with ventricular rate @ 140bpm (atrial flutter with 2:1 block)

- The ventricular response may be 2:1, 3:1 (rare), 4:1, or irregular depending upon the AV conduction properties and AV node slowing drugs on board (e.g., digoxin, beta blockers).

## 5. Ectopic Atrial Tachycardia and Rhythm

- Ectopic, discrete looking, unifocal P' waves with atrial rate <250/min (not to be confused with slow atrial flutter)
- Ectopic P' waves usually precede QRS complexes with P'R interval < RP' interval (i.e., not to be confused with paroxysmal supraventricular tachycardia with retrograde P waves appearing shortly after the QRS complexes).
- Ventricular response may be 1:1 or with varying degrees of AV block (especially in digitalis toxicity, as shown in this 3-lead ECG with 2:1 block).



[click here to view](#)

- Ectopic atrial rhythm is similar to ectopic atrial tachycardia, but with HR <100 bpm.

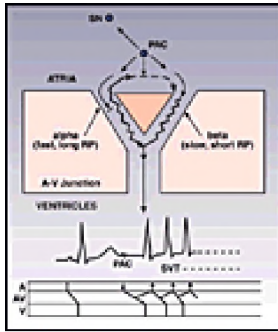
## 6. Multifocal Atrial Tachycardia (MAT) and rhythm

- Discrete, multifocal P' waves occurring at rates of 100-250/min and with varying P'R intervals (should see at least 3 different P wave morphologies in a given lead).
- Ventricular response is irregularly irregular (i.e., often confused with A-fib).
- May be intermittent, alternating with periods of normal sinus rhythm.
- Seen most often in elderly patients with chronic or acute medical problems such as exacerbation of chronic obstructive pulmonary disease.
- If atrial rate is <100 bpm, **call it multifocal atrial rhythm**

## 7. Paroxysmal Supraventricular Tachycardia (PSVT)

- **Basic Considerations:** These arrhythmias are **circus movement** or **reciprocating** tachycardias because they utilize the mechanism of **reentry**. The onset is sudden, usually initiated by a premature beat, and the arrhythmia also stops abruptly - which is why they are called **paroxysmal**. They are usually narrow-QRS tachycardias unless there is preexisting bundle branch block or rate-related aberrant ventricular conduction. There are several types of PSVT depending on the location of the reentry circuit.
- **AV Nodal Reentrant Tachycardia (AVNRT):** This is the most common form of PSVT accounting for approximately 50% of all symptomatic PSVTs. The diagram illustrates the probable mechanism involving dual AV nodal pathways, **alpha** and **beta**, with different electrical properties. In the diagram **alpha** is a fast AV nodal pathway with a long refractory period (RP), and **beta** is the slow pathway with a short RP. During sinus rhythm **alpha** is always used because it conducts faster. An early PAC, however, finds **alpha** still refractory and must use the slower **beta** pathway to reach the ventricles. By the time it traverses **beta**, however, **alpha** has recovered allowing retrograde conduction back to the atria. The

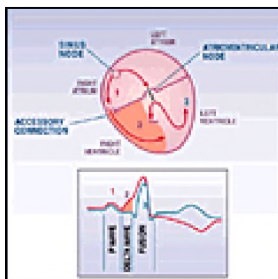
retrograde P wave (called an **atrial echo** for obvious reasons) is often simultaneous with the QRS and, therefore, not seen on the ECG, but it can reenter the AV junction because of **beta's** short RP.



[click here to view](#)

If conditions are right, a circus movement or reciprocating tachycardia results as seen in the above ECG and ladder diagram. Rarely, an "uncommon" form of AVNRT occurs with the retrograde P wave appearing in front of the next QRS (i.e., RP' interval  $> 1/2$  RR interval), implying antegrade conduction down the faster **alpha**, and retrograde conduction up the slower **beta**.

➤ **AV Reciprocating Tachycardia (Extranodal bypass pathway):** This is the second most common form of PSVT and is seen in patients with WPW syndrome. The WPW ECG, seen in the diagram, shows a short PR, **deltawave**, and somewhat widened QRS.



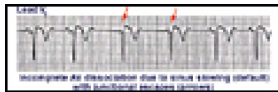
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This type of PSVT can also occur in the absence of manifest WPW on a preceding ECG if the accessory pathway only allows conduction in the retrograde direction (i.e., **concealed** WPW). Like AVNRT, a PAC that finds the bypass track temporarily refractory usually initiates the onset of PSVT. The PAC conducts down the normal AV pathway to the ventricles, and reenters the atria retrogradely through the bypass track. In this type of PSVT retrograde P waves appear shortly after the QRS in the ST segment (i.e., RP'  $< 1/2$  RR interval). Rarely the antegrade limb for PSVT uses the bypass track and the retrograde limb uses the AV junction; the PSVT then resembles a wide QRS tachycardia and must be differentiated from ventricular tachycardia.

➤ **Sino-Atrial Reentrant Tachycardia:** This is a rare form of PSVT where the reentrant circuit is between the sinus node and the right atria. The ECG looks like sinus tachycardia, but the tachycardia is paroxysmal; i.e., it starts and ends abruptly.

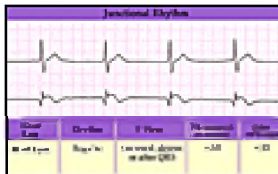
## 8. Junctional Rhythms and Tachycardias

➤ **Junctional Escape Beats:** These are *passive*, protective beats originating from subsidiary pacemaker cells in the AV junction (usually in the Bundle of His). The pacemaker's basic firing rate is 40-60 bpm; junctional escapes are protective events that occur whenever the primary pacemaker (i.e., sinus node) defaults or the AV node blocks the atrial impulse. The ECG strip shows intermittent sinus slowing with two junctional escapes.



[click here to view](#)

➤ **Junctional Escape Rhythm:** This is a sequence of 3 or more junctional escapes occurring by default at a rate of 40-60 bpm. There may be AV dissociation or the atria may be captured retrogradely by the junctional pacemaker. In the ECG example below the retrograde P waves are not seen and must be hidden in the QRS's; the significant "Q" wave with ST elevation in the bottom strip suggests an acute MI.



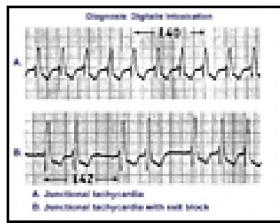
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➤ **Accelerated Junctional Rhythm:** This is an *active* junctional pacemaker rhythm caused by events that perturb pacemaker cells (e.g., ischemia, drugs, and electrolyte abnormalities). The rate is 60-100 bpm).



[click here to view](#)

➤ **Nonparoxysmal Junctional Tachycardia:** This usually begins as an accelerated junctional rhythm but the heart rate gradually increases to >100 bpm. There may be AV dissociation, or retrograde atrial capture may occur. Ischemia (usually from right coronary artery occlusion) and digitalis intoxication are the two most common causes. In the example below junctional tachycardia is seen with ('B') and without exit block ('A').



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 [Return to the beginning of Lesson V](#)

 [Move on to Ventricular Arrhythmias](#)



## Lesson V (cont) Ventricular Arrhythmias

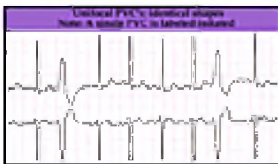
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I II III IV **V** VI VII VIII IX X XI XII

[Return to the beginning of Lesson V](#)

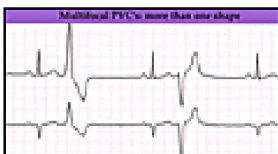
1. [Premature ventricular complexes \(PVCs\)](#)
2. [Aberrancy vs. ventricular ectopy](#)
3. [Ventricular tachycardia](#)
4. [Differential diagnosis of wide QRS tachycardias](#)
5. [Accelerated ventricular rhythms](#)
6. [Idioventricular rhythm](#)
7. [Ventricular Parasystole](#)

### 1. Premature Ventricular Complexes (PVCs)



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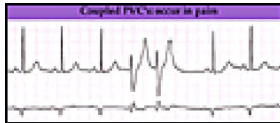
➤ PVCs may be unifocal(see above), multifocal(see below) or multiformed. Multifocal PVCs have different sites of origin, which means their coupling intervals (measured from the previous QRS complexes) are usually different. Multiformed PVCs usually have the same coupling intervals (because they originate in the same ectopic site but their conduction through the ventricles differ. Multiformed PVCs are common in digitalis intoxication.



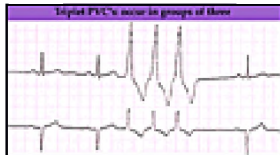
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➤ PVCs may occur as isolated single events or as couplets, triplets, and salvos (4-6 PVCs in a row), also called brief ventricular tachycardias.

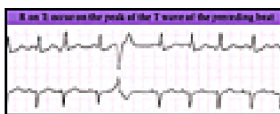


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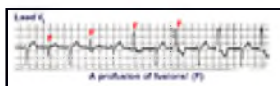


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➤ PVCs may occur early in the cycle (R-on-T phenomenon), after the T wave (as seen above), or late in the cycle - often fusing with the next QRS (fusion beat). R-on-T PVCs may be especially dangerous in an acute ischemic situation, because the ventricles may be more vulnerable to ventricular tachycardia or fibrillation. Examples are seen below.



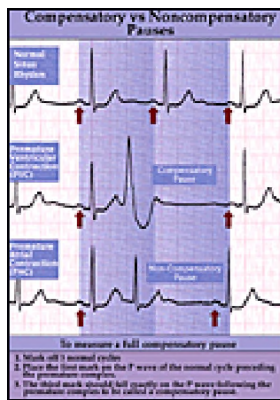
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In the above example, "late" (end-diastolic) PVCs are illustrated with varying degrees of fusion. For fusion to occur the sinus P wave must have made it to the ventricles to start the activation sequence, but before ventricular activation is completed the "late" PVC occurs. The resultant QRS looks a bit like the normal QRS, and a bit like the PVC; i.e., a fusion QRS.

➤ The events following a PVC are of interest. Usually a PVC is followed by a complete compensatory pause because the sinus node timing is not interrupted; one sinus P wave isn't able to reach the ventricles because they are still refractory from the PVC; the following sinus impulse occurs on time based on the sinus rate. In contrast, PACs are usually followed by an incomplete pause because the PAC usually enters the sinus node and resets its timing; this enables the following sinus P wave to appear earlier than expected. These concepts are illustrated below.



[click here to view](#)

➤ Not all PVCs are followed by a pause. If a PVC occurs early enough (especially if the heart rate is slow), it may appear sandwiched in between two normal beats. This is called an interpolated PVC. The sinus impulse following the PVC may be conducted with a longer PR interval because of retrograde concealed conduction by the PVC into the AV junction slowing subsequent conduction of the sinus impulse.



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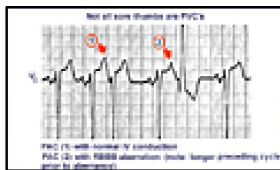
➤ Finally a PVC may retrogradely capture the atrium, reset the sinus node, and be followed by an incomplete pause. Often the retrograde P wave can be seen on the ECG, hiding in the ST-T wave of the PVC.

➤ The most unusual post-PVC event is when retrograde activation of the AV junction re-enters the ventricles as a ventricular echo. This is illustrated below. The "ladder" diagram below the ECG helps us understand the mechanism. The P wave following the PVC is the sinus P wave, but the PR interval is too short for it to have caused the next QRS. (Remember, the PR interval following an interpolated PVC is usually longer than normal, not shorter!).



[click here to view](#)

➤ PVCs usually stick out like "sore thumbs", because they are bizarre in appearance compared to the normal complexes. However, not all premature sore thumbs are PVCs. In the example below 2 PACs are seen, #1 with a normal QRS, and #2 with RBBB aberrancy - which looks like a sore thumb. **The challenge, therefore, is to recognize sore thumbs for what they are, and that's the next topic for discussion!**



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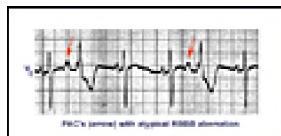
## 2. Aberrancy vs. Ventricular Ectopy

A most important question

➤ **Aberrant Ventricular Conduction:** defined as the intermittent abnormal intraventricular conduction of a supraventricular impulse. The phenomenon comes about because of unequal refractoriness of the bundle branches and critical prematurity of a supraventricular impulse (see [diagram](#) of "Three Fates of PACs"). With such critical prematurity, the supraventricular impulse encounters one bundle branch (or fascicle) which is responsive, and the other which is refractory, and is consequently conducted with a bundle branch block or fascicular block pattern.

➤ ECG clues to the differential diagnosis of wide QRS premature beats:

● Preceding ectopic P wave (i.e., the P' of the PAC) usually hidden in the ST-T wave of the previous beat favors aberrant ventricular conduction. In the ECG below note the arrow pointing at a premature P wave in the ST-T segment. The QRS has a RBBB morphology.



[click here to view](#)

● Analyze the compensatory pause: A complete pause favors ventricular ectopy (i.e., no resetting of the sinus pacemaker; next sinus impulse comes on time). An incomplete pause favors aberration (i.e., because supraventricular prematures are more likely to reset the sinus node's timing). Be aware of exceptions to this simple rule because PVCs can activate the atria retrogradely and reset the sinus node (incomplete pause), and PACs can fail to reset the sinus node (complete pause).

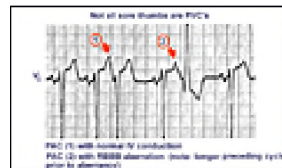
● Long-Short Rule (Ashman Phenomenon): The earlier in the cycle a PAC occurs and the longer the preceding cycle, the more likely the PAC will be conducted with aberration (see [diagram](#) "The Three Fates of PACs"). This is because the refractory period of the ventricular conduction system is proportional to cycle length or heart rate; the longer the cycle length or slower the heart rate, the longer the recovery time of the conduction system. In most individuals the right bundle normally recovers more slowly than the left bundle, and a critically

timed PAC is therefore more likely to conduct with RBBB than with LBBB. In diseased hearts, however, LBBB aberrancy is also seen. Dr. Richard Ashman and colleagues first described this in 1947 in patients with atrial fibrillation. He noted that the QRS complexes ending a short RR interval were often of a RBBB pattern if the preceding RR interval was long. (That's all it takes to get your name attached to a phenomenon; you must publish!).

● Analyze the **QRS morphology** of the funny-looking beat. This is one of the most rewarding of the clinical clues, especially if lead V1 (or the MCL1 monitored lead in intensive care units) is used. Since aberrancy is almost always in the form of a bundle branch block morphology, V1 is the best lead for differentiating RBBB from LBBB; RBBB creates a positive deflection, and LBBB, a negative deflection. Therefore, the first order of business is to identify the direction of QRS forces in V1.

If the QRS in V1 is mostly positive the following possibilities exist:

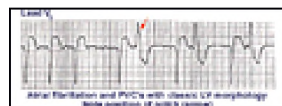
■ rsR' or rSR' QRS morphologies suggests RBBB aberrancy >90% of the time!



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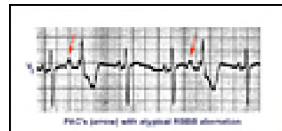
Note the rsR' morphology of PAC #2!

■ monophasic R waves or R waves with a notch or slur on the downstroke of the R waves suggests ventricular ectopy > 90% of the time (see below)!



[click here to view](#)

■ monophasic R wave with a notch or slur on the upstroke of R wave: 50-50 possibility or either!



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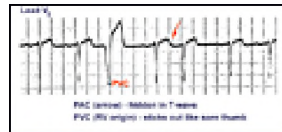
In the above ECG the premature wide QRS is an aberrantly conducted PAC because of the easily seen preceding P wave. The QRS morphology could be either!

■ qR morphology suggests ventricular ectopy unless a previous anteroseptal MI or unless the patient's normal V1 QRS complex has a QS morphology (i.e., no initial r-wave)!

If the QRS in V1 is mostly negative the following possibilities exist:

■ Rapid downstroke of the S wave with or without a preceding "thin" r wave suggests LBBB aberrancy almost always!

■ Fat" r wave (0.04s) or notch/slur on downstroke of S wave or >0.06s delay from QRS onset to nadir of S wave almost always suggests ventricular ectopy!



[click here to view](#)

In the above ECG the wide premature QRS is a PVC because of the >0.06s delay from onset of the QRS to the nadir of the S wave (approximately 0.08s).

Another QRS morphology clue from Lead V6:

■ If the wide QRS morphology is predominately negative in direction in lead V6, then it's most likely ventricular ectopy (assuming V6 is accurately placed in mid axillary line)!

● The timing of the premature wide QRS complex is also important because aberrantly conducted QRS complexes only occur early in the cardiac cycle during the refractory period of one of the conduction branches. Therefore, late premature wide QRS complexes (after the T wave, for example) are most often ventricular ectopic in origin.

### 3. Ventricular Tachycardia

➤ Descriptors to consider when considering ventricular tachycardia:

● Sustained (lasting >30 sec) vs. nonsustained

● Monomorphic (uniform morphology) vs. polymorphic vs. Torsade-de-pointes

■ **Torsade-de-pointes:** a polymorphic ventricular tachycardia associated with the long-QT syndromes characterized by phasic variations in the polarity of the QRS complexes around the baseline. Ventricular rate is often >200bpm and ventricular fibrillation is a consequence.

● Presence of AV dissociation (independent atrial activity) vs. retrograde atrial capture

● Presence of fusion QRS complexes (Dressler beats) which occur when supraventricular beats (usually sinus) get into the ventricles during the ectopic activation sequence.

➤ **Differential Diagnosis:** just as for single premature funny-looking beats, **not all wide QRS tachycardias are ventricular in origin (i.e., they may be supraventricular tachycardias with bundle branch block or WPW preexcitation)!**

## 4. Differential Diagnosis of Wide QRS Tachycardias

➤ Although this is an ECG tutorial, let's not forget some simple bedside clues to ventricular tachycardia:

● Advanced heart disease (e.g., coronary heart disease) statistically favors ventricular tachycardia

● Cannon 'a' waves in the jugular venous pulse suggests ventricular tachycardia with AV dissociation. Under these circumstances atrial contractions may occur when the tricuspid valve is still closed which leads to the giant retrograde pulsations seen in the JV pulse. With AV dissociation these giant a-waves occur irregularly.

● Variable intensity of the S1 heart sound at the apex (mitral closure); again this is seen when there is AV dissociation resulting in varying position of the mitral valve leaflets depending on the timing of atrial and ventricular systole.

● If the patient is hemodynamically unstable, **think ventricular tachycardia** and act accordingly!

➤ ECG Clues:

● Regularity of the rhythm: If the wide QRS tachycardia is sustained and monomorphic, then the rhythm is usually regular (i.e., RR intervals equal); an irregularly-irregular rhythm suggests atrial fibrillation with aberration or with WPW preexcitation.

● A-V Dissociation strongly suggests ventricular tachycardia! Unfortunately AV

dissociation only occurs in approximately 50% of ventricular tachycardias (the other 50% have retrograde atrial capture or "V-A association"). Of the patients with AV dissociation, it is only easily recognized if the rate of tachycardia is <150 bpm. Faster heart rates make it difficult to visualize dissociated P waves.

● Fusion beats or captures often occur when there is AV dissociation and this also strongly suggests a ventricular origin for the wide QRS tachycardia.

● QRS morphology in lead V1 or V6 as described above for single premature funny looking beats is often the best clue to the origin, so go back and check out the clues! Also consider a few other morphology clues:

■ Bizarre frontal-plane QRS axis (i.e. from +150 degrees to -90 degrees or NW quadrant) suggests ventricular tachycardia

■ QRS morphology similar to previously seen PVCs suggests ventricular tachycardia

■ If all the QRS complexes from V1 to V6 are in the same direction (positive or negative), ventricular tachycardia is likely

■ Especially wide QRS complexes (>0.16s) suggests ventricular tachycardia

■ Also consider the following **Four-step Algorithm** reported by *Brugada et al, Circulation 1991;83:1649*:

**Step 1:** Absence of RS complex in all leads V1-V6?  
**Yes: Dx is ventricular tachycardia!**

**Step 2: No:** Is interval from beginning of R wave to nadir of S wave >0.1s in any RS lead?  
**Yes: Dx is ventricular tachycardia!**

**Step 3: No:** Are AV dissociation, fusions, or captures seen?  
**Yes: Dx is ventricular tachycardia!**

**Step 4: No:** Are there morphology criteria for VT present both in leads V1 and V6?  
**Yes: Dx is ventricular tachycardia!**

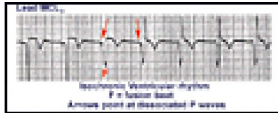
**NO: Diagnosis is supraventricular tachycardia with aberration!**

## 5. Accelerated Ventricular Rhythms

(see ECG below)



- An "active" ventricular rhythm due to enhanced automaticity of a ventricular pacemaker (reperfusion after thrombolytic therapy is a common causal factor).
- Ventricular rate 60-100 bpm (anything faster would be ventricular tachycardia)
- Sometimes called ***isochronic ventricular rhythm*** because the ventricular rate is close to underlying sinus rate
- May begin and end with fusion beats (ventricular activation partly due to the normal sinus activation of the ventricles and partly from the ectopic focus).
- Usually benign, short lasting, and not requiring of therapy.



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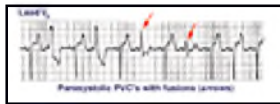
## 6. Idioventricular Rhythm

- A "passive" escape rhythm that occurs by default whenever higher-level pacemakers in AV junction or sinus node fail to control ventricular activation.
  - Escape rate is usually 30-50 bpm (i.e., slower than a junctional escape rhythm).
  - Seen most often in complete AV block with AV dissociation or in other bradycardic conditions.

## 7. Ventricular Parasystole

- Non-fixed coupled PVCs where the inter-ectopic intervals (i.e., timing between PVCs) are some multiple (i.e., 1x, 2x, 3x, . . . etc.) of the basic rate of the parasystolic focus
- PVCs have uniform morphology unless fusion beats occur
- Usually entrance block is present around the ectopic focus, which means that the primary rhythm (e.g., sinus rhythm) is unable to enter the ectopic site and reset its timing.
- May also see exit block; i.e., the output from the ectopic site may occasionally be blocked (i.e., no PVC when one is expected).
- Fusion beats are common when ectopic site fires while ventricles are already being activated from primary pacemaker





[click here to view](#)

➤ Parasystolic rhythms may also be seen in the atria and AV junction



◀ [Return to the beginning of Lesson V](#)

▶ [Go back to supraventricular arrhythmias](#)



## Lesson II (cont) How to Measure the QRS Axis

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[I](#) [II](#) [III](#) [IV](#) [V](#) [VI](#) [VII](#) [VIII](#) [IX](#) [X](#) [XI](#) [XII](#)

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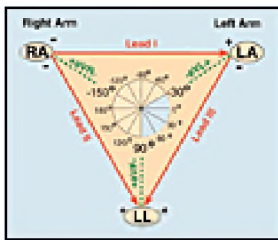
- 
1. [Introduction](#)
  2. [QRS Axis Determination](#)
  3. [Examples of QRS Axis](#)

### 1. Introduction

The frontal plane QRS axis represents only the average direction of ventricular activation in the frontal plane. As such this measure can inform the ECG reader of changes in the sequence of ventricular activation (e.g., left anterior fascicular block), or it can be an indicator of myocardial damage (e.g., inferior myocardial infarction).

In the diagram below the normal range is identified ( $-30^{\circ}$  to  $+90^{\circ}$ ). Left axis deviation (i.e., superior and leftward) is defined from  $-30^{\circ}$  to  $-90^{\circ}$ , and right axis deviation (i.e., inferior and rightward) is defined from  $+90^{\circ}$  to  $+150^{\circ}$ .

Click to see [causes of abnormal axis \(lesson 4\)](#).



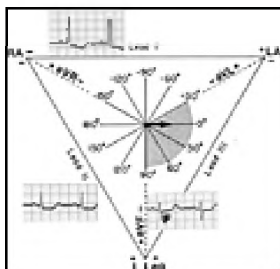
[Click to view](#) (Requires an Internet connection)

## 2. QRS Axis Determination

- First find the *isoelectric* lead if there is one; i.e., the lead with equal forces in the positive and negative direction. Often this is the lead with the smallest QRS.
- The QRS axis is *perpendicular* to that lead's orientation (see above diagram).
- Since there are two perpendiculars to each isoelectric lead, chose the perpendicular that best fits the direction of the other ECG leads.
- If there is no isoelectric lead, there are usually *two* leads that are nearly isoelectric, and these are always 30° apart. Find the perpendiculars for each lead and chose an approximate QRS axis within the 30° range.
- Occasionally each of the 6 frontal plane leads is small and/or isoelectric. The axis cannot be determined and is called *indeterminate*. This is a normal variant.

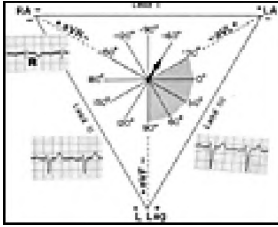
## 3. Examples of QRS Axis

- Axis in the normal range:



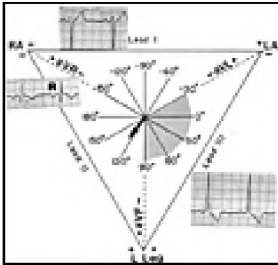
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➤ Axis in the left axis deviation(LAD) range:



[Click to view](#)

➤ Axis in the right axis deviation(RAD) range:

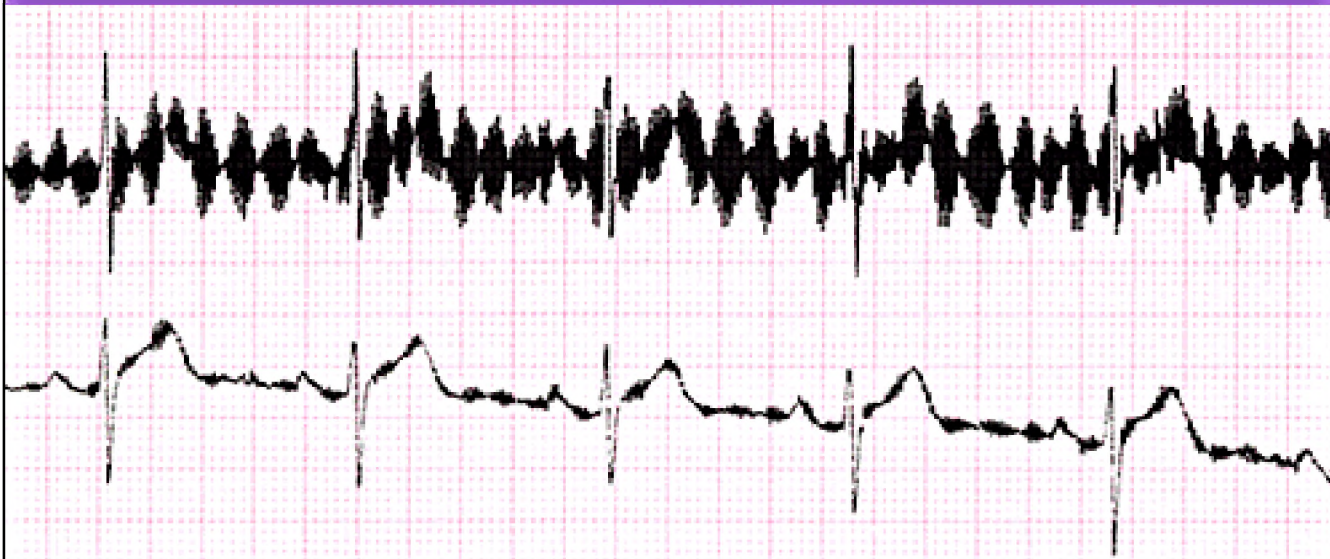


[Click to view](#)

[Return to the beginning of Lesson II](#)



## AC Interference (60 cycle)



**Sixty even, regular spikes in a 1 second interval caused by electrical current near the patient**

## 60 Cycle Artifact - Marquette-KH

*Frank Yanowitz Copyright 1996*

## Wandering Baseline (drift)



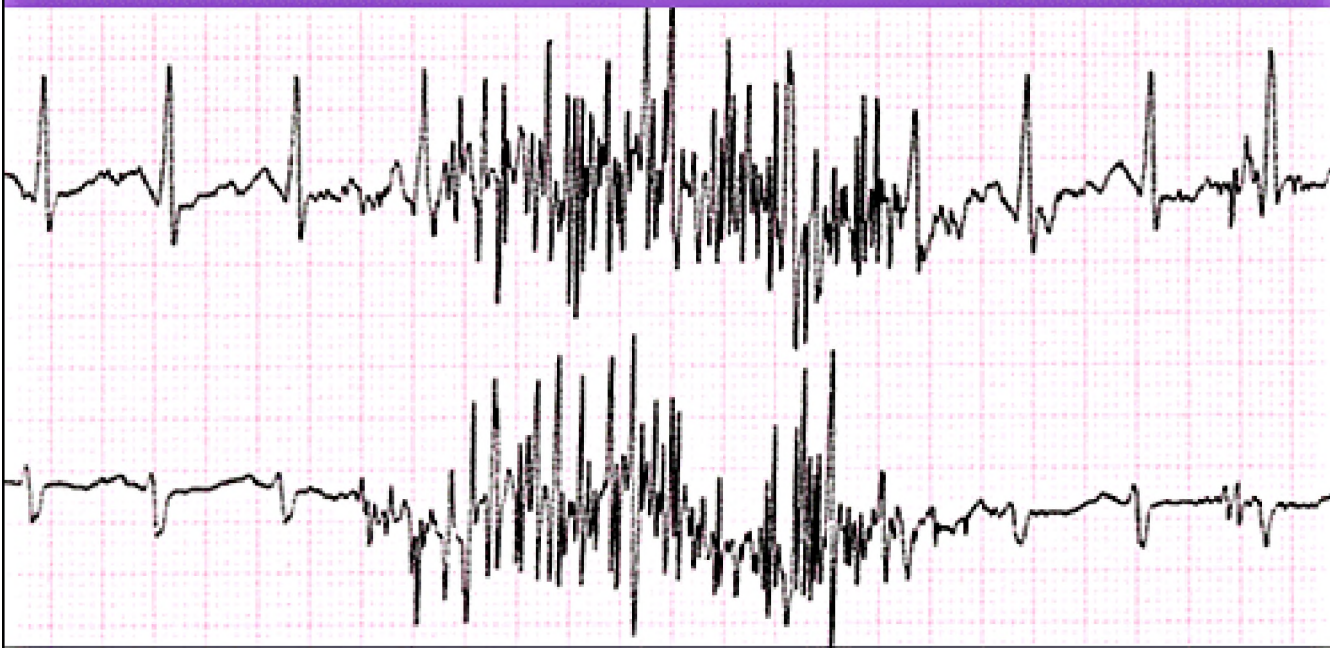
**An undulating baseline with waveform present.**

## Wandering Baseline Artifact - Marquette-KH

*Marquette Electronics Copyright 1996*



## Muscle Tremor (somatic)

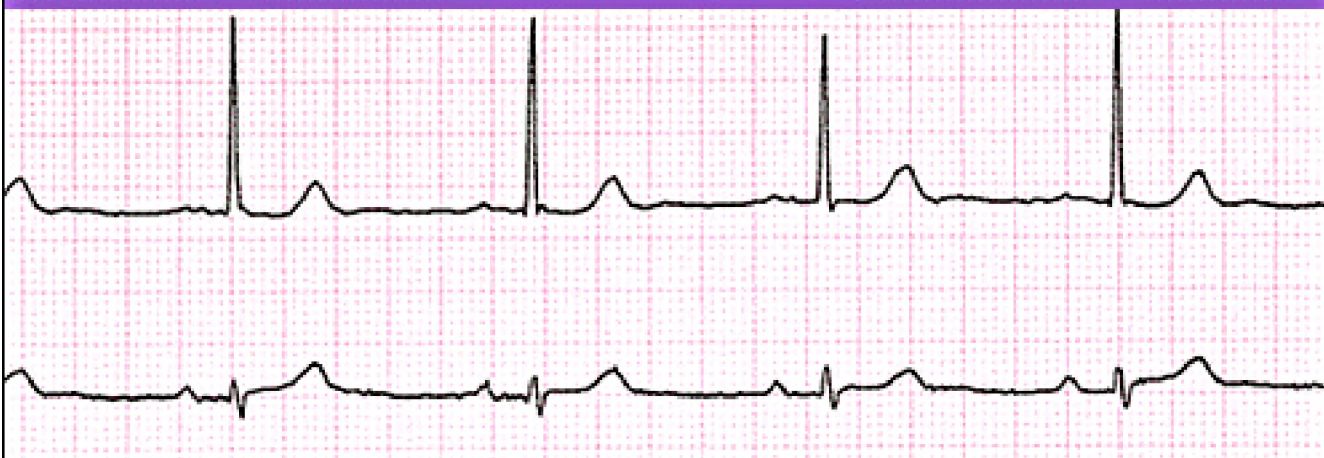


**Electrical interference caused by the patient's tensed muscles.**

## Muscle Tremor Artifact - Marquette-KH

*Marquette Electronics Copyright 1996*

## Sinus Bradycardia



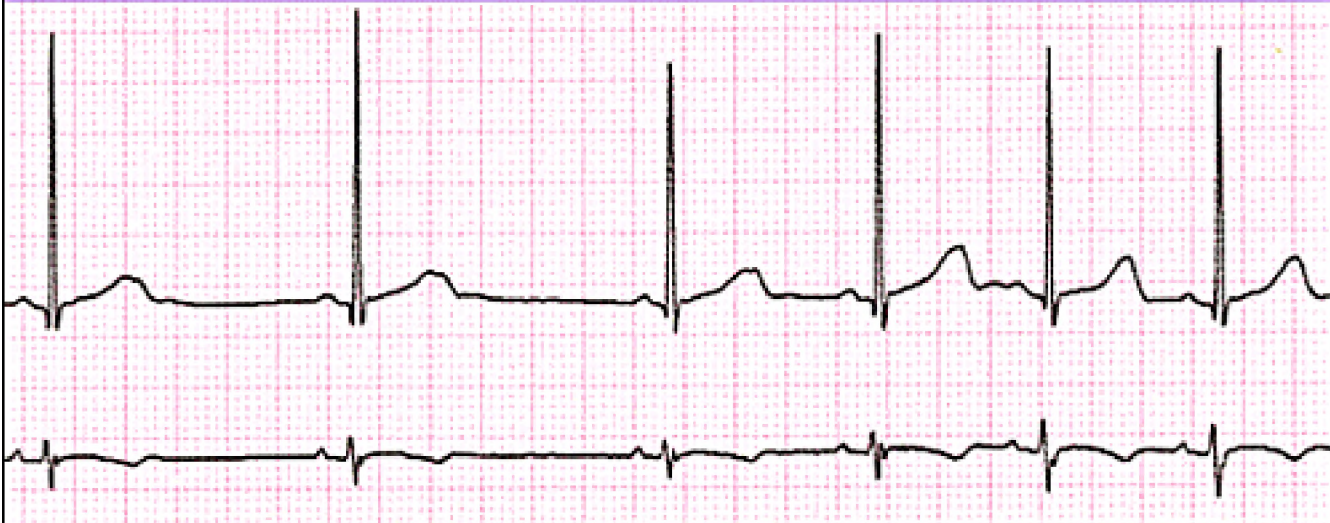
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
< 60 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

## Sinus Bradycardia-KH

*Frank G. Yanowitz, M.D., copyright 1997*



## Sinus Arrhythmia

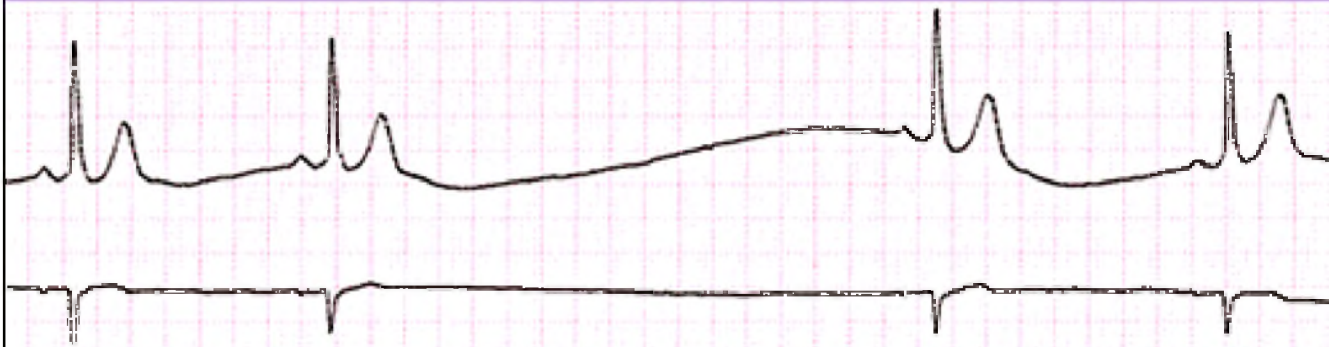


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
Usually 60-100 bpm	Irregular	Before each QRS, identical	.12 to .20	<.12

## Marked Sinus Arrhythmia - Marquette-KH

*Marquette Electronics Copyright 1996*

## Sinus Arrest



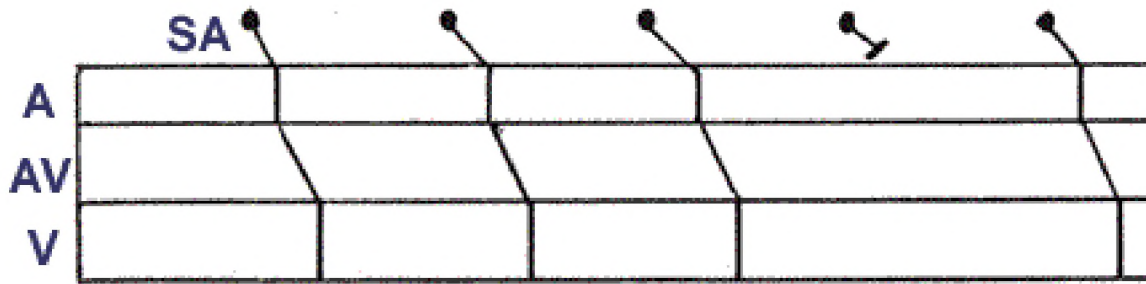
Please note: trace and grid have been reduced in size

Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Before each QRS identical. New rhythm begins after a pause. The P to P interval is disturbed.	.12 to .20	<.12

## Sinus Pause or Arrest - Marquette-KH

*Marquette Electronics Copyright 1996*

## Lead II



**Sino-Atrial Exit Block (type I)**

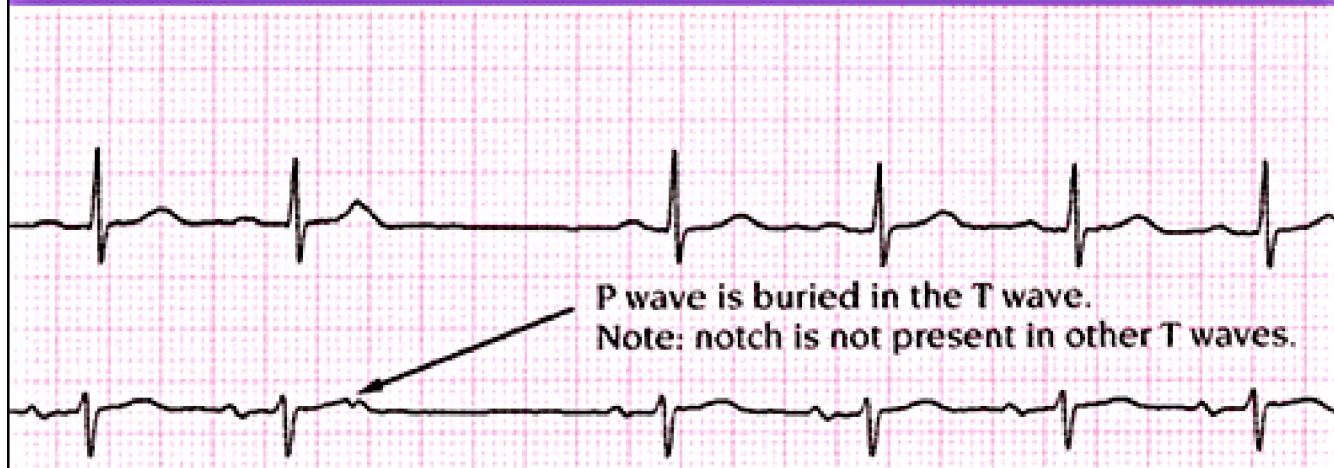
## Sino-Atrial Exit Block, Type I or Wenckebach-KH

*Frank Yanowitz Copyright 1996*

This example illustrates 2nd degree sino-atrial exit block. In type 1 S-A block the conduction time between sinus firing and atrial capture progressively prolong, but this cannot be seen on the ECG tracing; type I exit block is inferred if the P-P intervals gradually shorten before the pause and if the P-P interval of the pause is less than the two preceding P-P intervals. In type II S-A block the P-P interval of the pause is twice the basic P-P interval.



## Nonconducted Premature Atrial Contraction

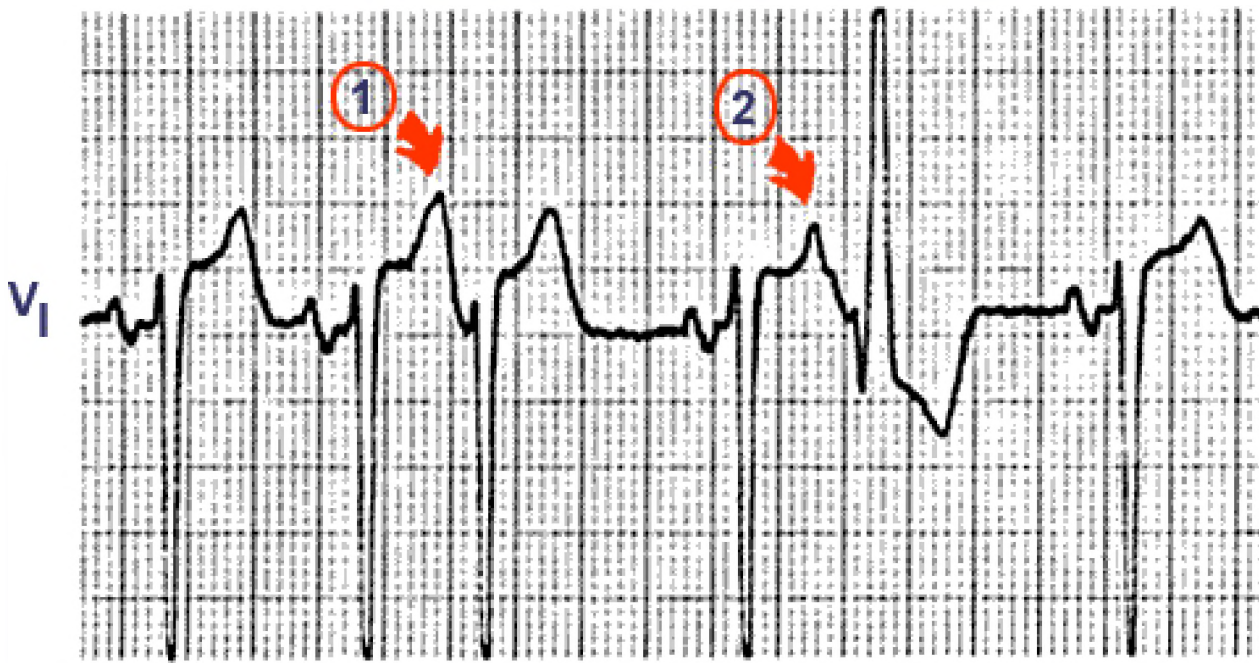


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Premature & abnormal or hidden	N/A	Absent

## Nonconducted PAC - Marquette-KH

*Marquette Electronics Copyright 1996*

## Not all sore thumbs are PVC's



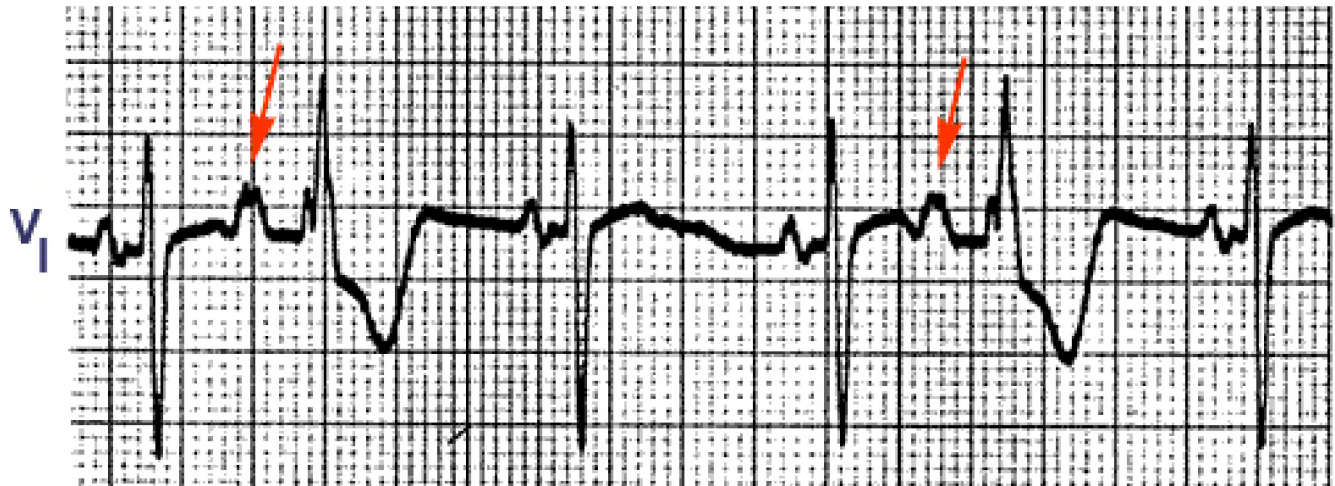
**PAC (1) with normal IV conduction**

**PAC (2) with RBBB aberration (note: longer preceding cycle prior to aberrancy)**

## Not All Sore Thumbs Are Ventricular In Origin-KH

*Frank Yanowitz Copyright 1996*

PAC's have three fates: normal conduction into ventricles, aberrant conduction in ventricles due to bundle branch or fascicular block, and non-conduction due to block in AV junction. In this example PAC '1' is normally conducted and PAC '2' is conducted with RBBB aberration. The longer preceding cycle increases the refractory period in the right bundle.

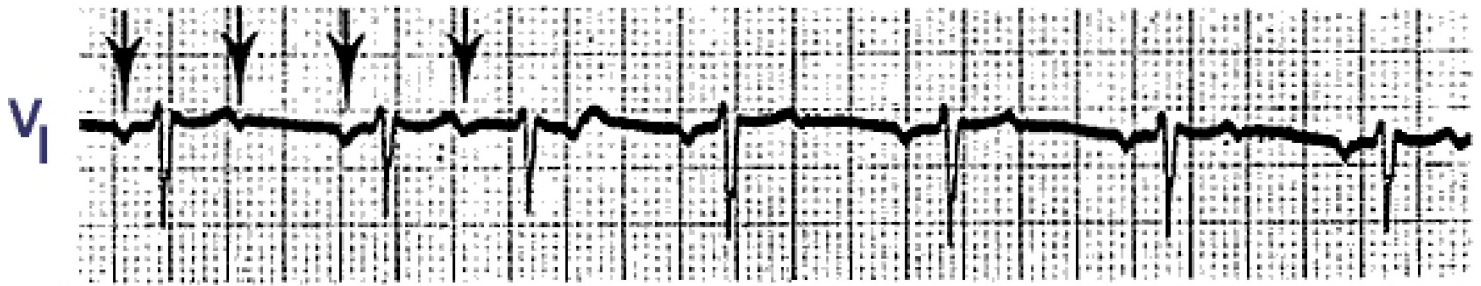


**PAC's (arrow) with atypical RBBB aberration**

## **PAC's With RBBB Aberration-KH**

*Frank Yanowitz Copyright 1996*

These PAC's, indicated by arrows, enter the ventricles and find the right bundle refractory. They therefore conduct with RBBB aberrancy. In most normal hearts the right bundle recovery time is longer than the left bundle's; most aberrancy, therefore, has a RBBB morphology. In some diseased hearts the left bundle may have a longer refractory period resulting in LBBB aberration. Aberrant conduction may also involve the fascicles of the left bundle.



**Diagnosis?**

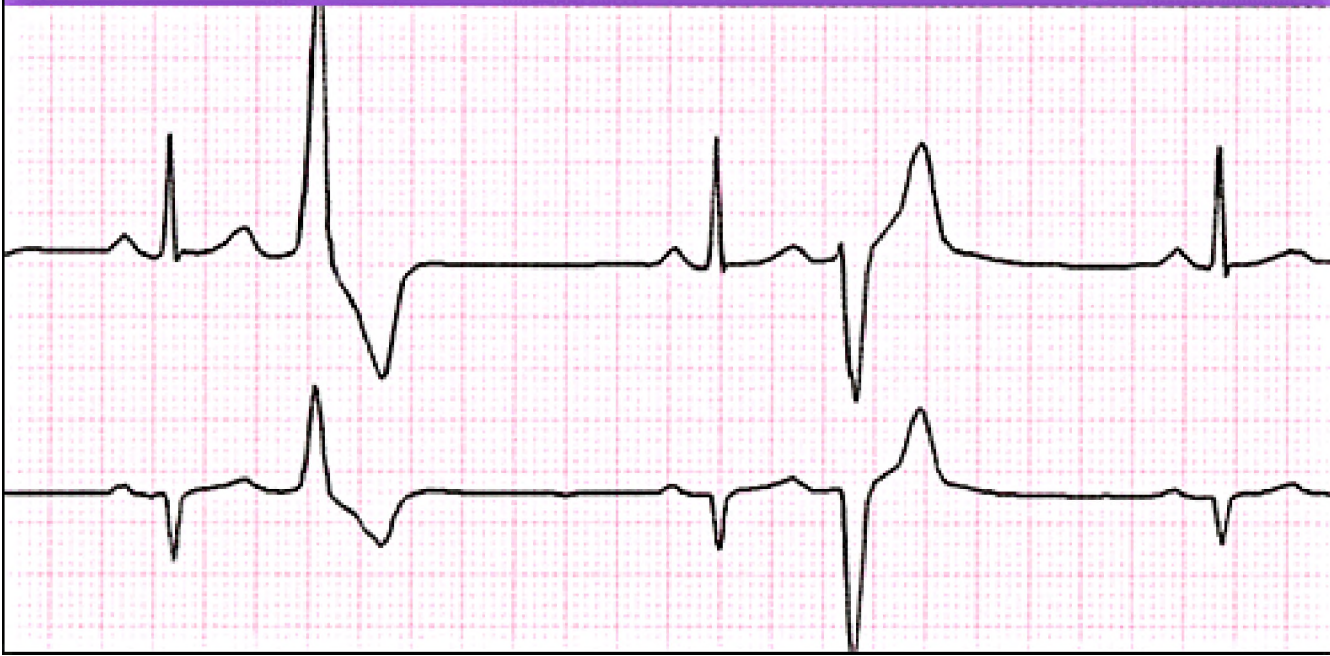
## **Atrial Tachycardia With 3:2 and 2:1 AV Block-KH**

*Frank Yanowitz Copyright 1996*

The ectopic atrial rate is 150 bpm. Some of the ectopic P waves are easily seen and indicated by the arrows. Other P waves are buried in the T waves and not so easily identified. Atrial tachycardia with AV block is often a sign of digitalis intoxication. 3:2 and 2:1 AV block is seen in this example.



## Multifocal PVC's: more than one shape



## Multifocal PVC's - Marquette-KH

*Marquette Electronics Copyright 1996*



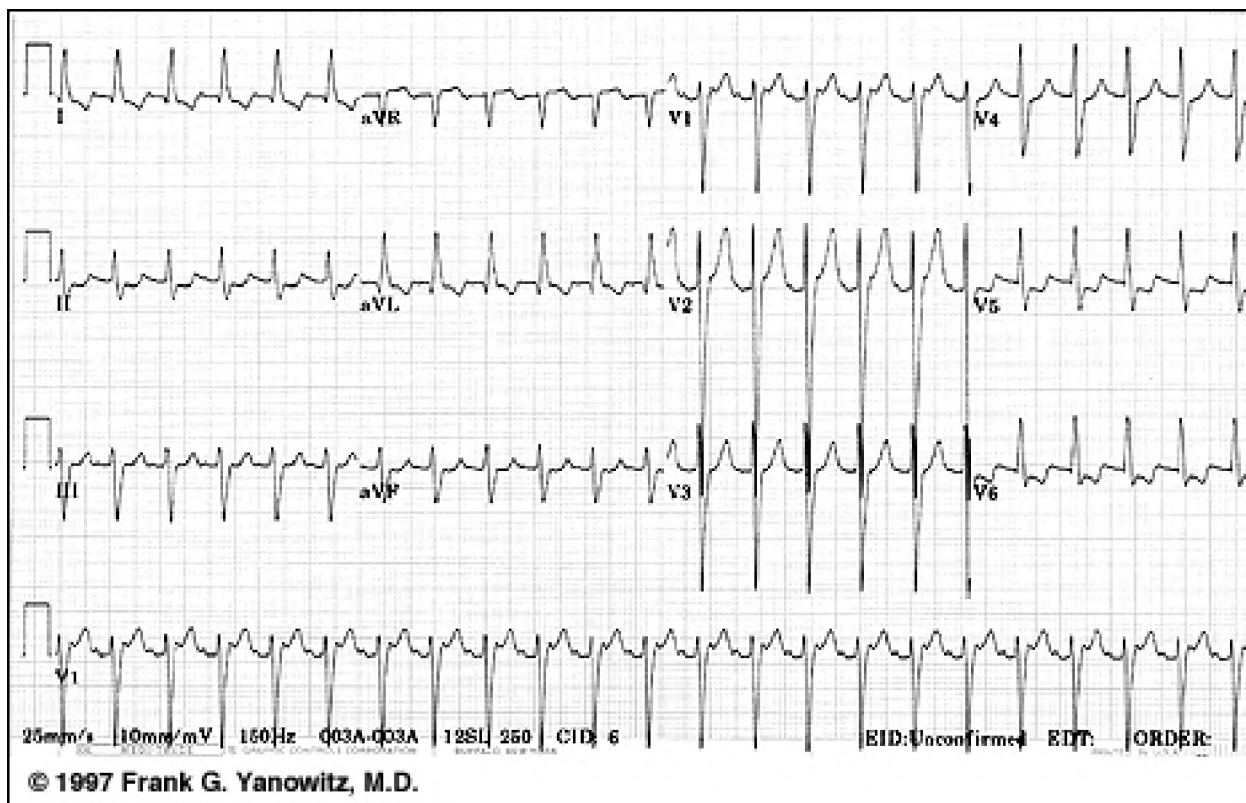
## Ventricular Pacemaker (single chamber)



One spike producing a wide QRS (ventricular capture).

## Ventricular Pacing in Atrial Fibrillation - Marquette-KH

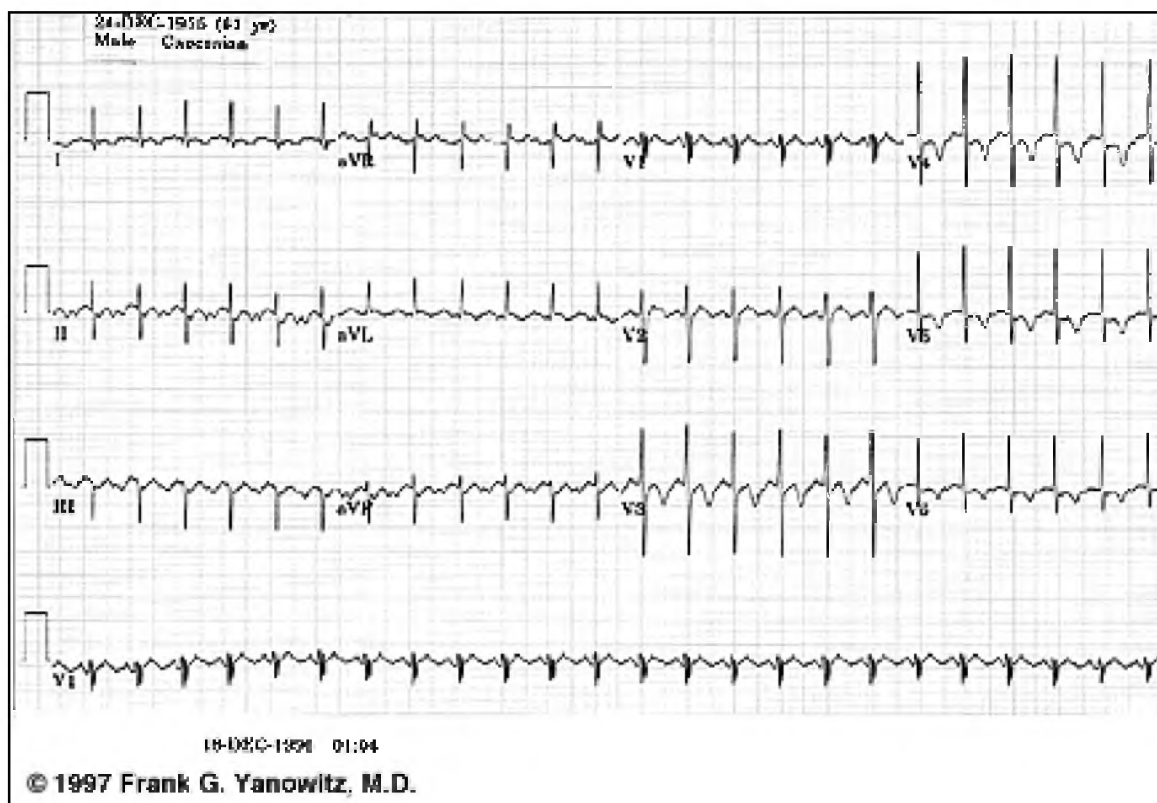
*Marquette Electronics Copyright 1996*



## Atrial Flutter With 2:1 AV Conduction-KH

*Frank G. Yanowitz, M.D.*

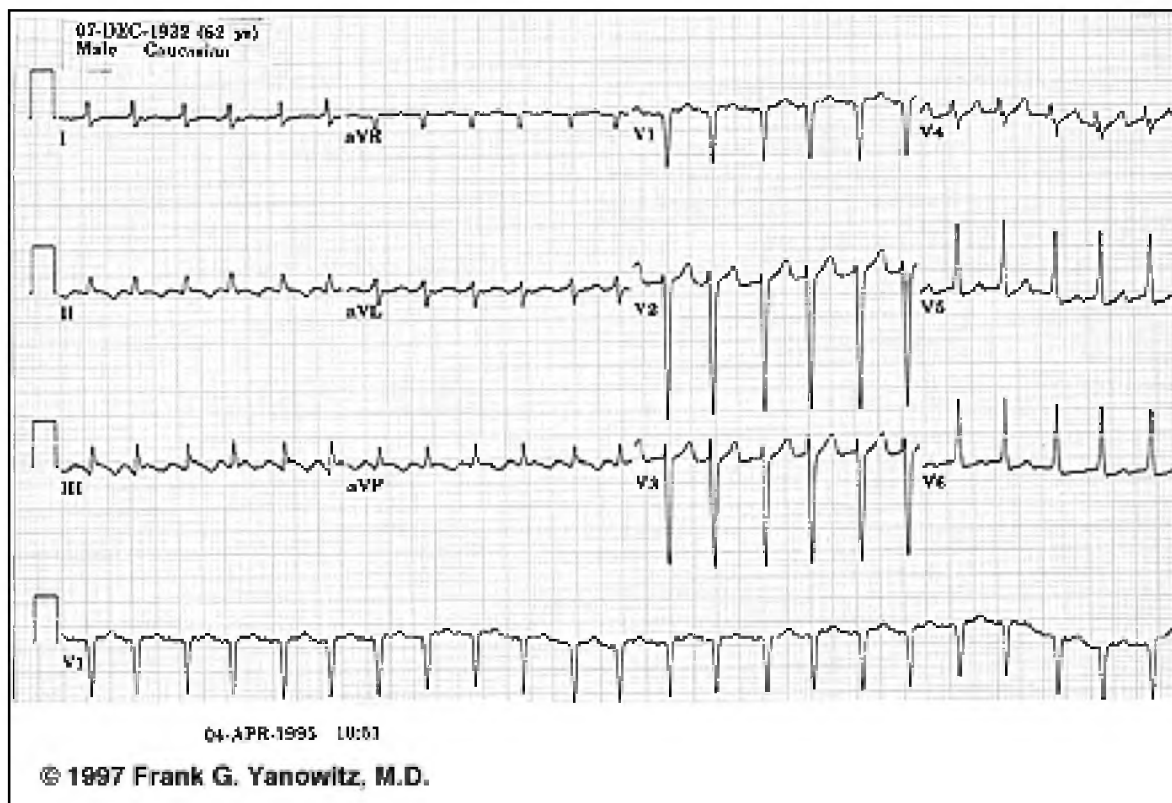
In this example of atrial flutter with 2:1 AV conduction the flutter waves are very hard to see. Atrial flutter with 2:1 block must be considered, however, because the heart rate is about 150 bpm. A careful look at V1 shows the two flutter waves for each QRS complex. One flutter wave immediately follows the QRS and the other is just before the QRS.



## Atrial Flutter With 2:1 AV Conduction-KH

*Frank G. Yanowitz, M.D.*

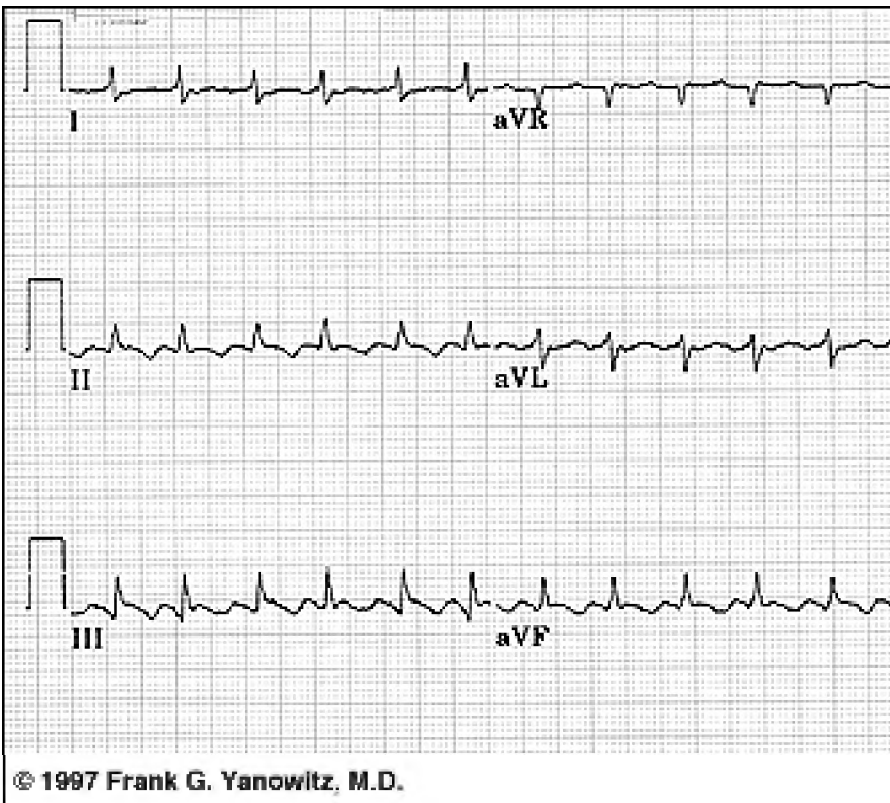
Atrial flutter with 2:1 AV block is one of the most frequently missed ECG rhythm diagnoses because the flutter waves are often hard to find. In this example two flutter waves for each QRS are best seen in lead III and V1. The ventricular rate at 150 bpm should always prompt us to consider atrial flutter with 2:1 conduction as a diagnostic consideration.



## Atrial Flutter With 3:2 AV Conduction-KH

*Frank G. Yanowitz, M.D.*

This 12-lead ECG shows a subtle bigeminal rhythm resulting from atrial flutter with a 3:2 AV conduction ratio; RR intervals alternate by a small duration. This is uncommon! The impulses from the atrial flutter conduct through the AV junction in a Wenckebach sequence; for every 3 flutter waves the second conducts more slowly than the first, and the third flutter wave is blocked.



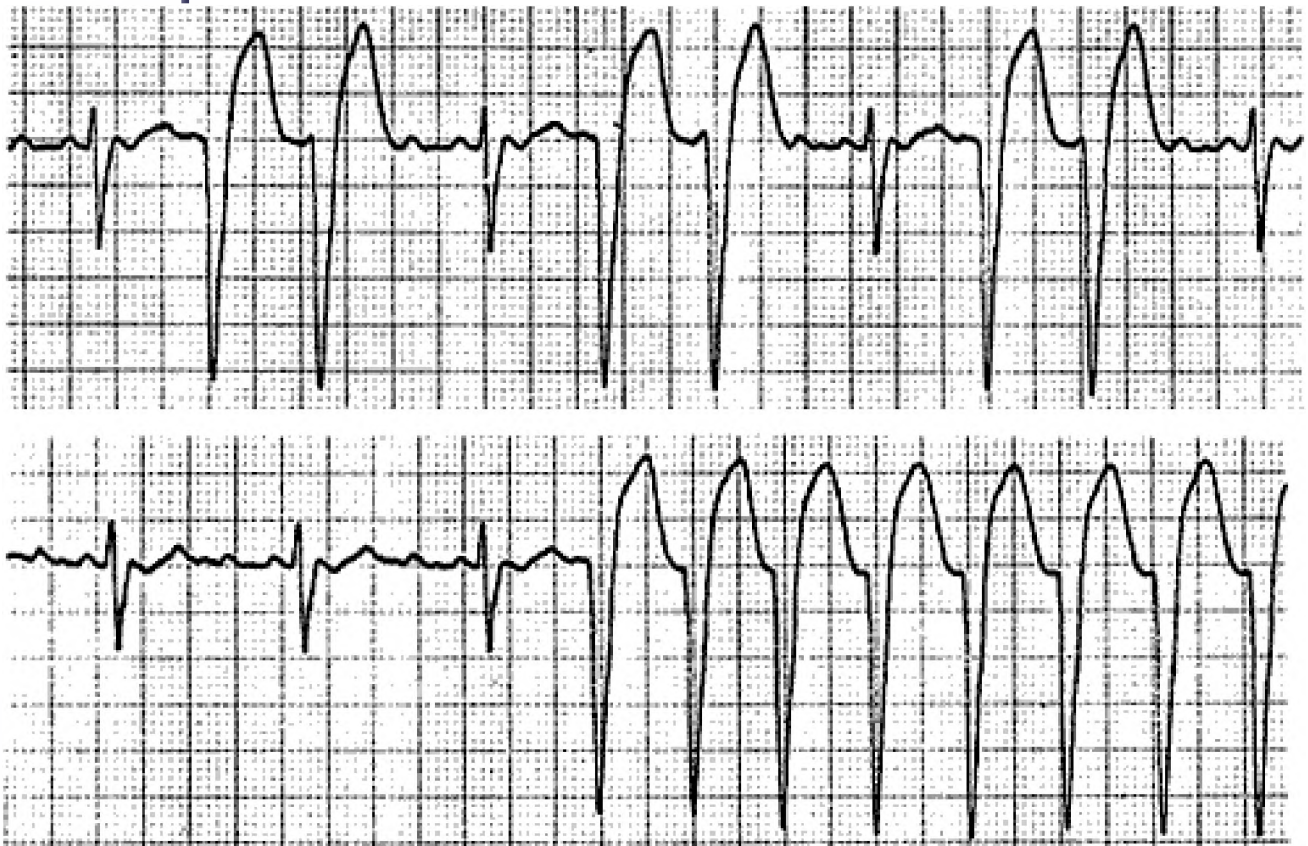
## Atrial Flutter with 3:2 Conduction Ratio: Frontal Plane Leads-KH

*Frank G. Yanowitz, M.D.*

Note the subtle bigeminy in the RR intervals. The best way to identify the flutter waves in this example is to imagine what lead III would look like if the QRS complexes disappeared; what remains is a reasonable "saw-tooth" pattern characteristic of atrial flutter with a flutter rate of about 300 bpm.



## Lead V<sub>1</sub>



**Question:**

- 1) What's the rhythm?
- 2) Does the patient need Lidocaine?

## Atrial Flutter With Variable AV Block And Rate-Dependent LBBB-KH

*Frank Yanowitz Copyright 1996*

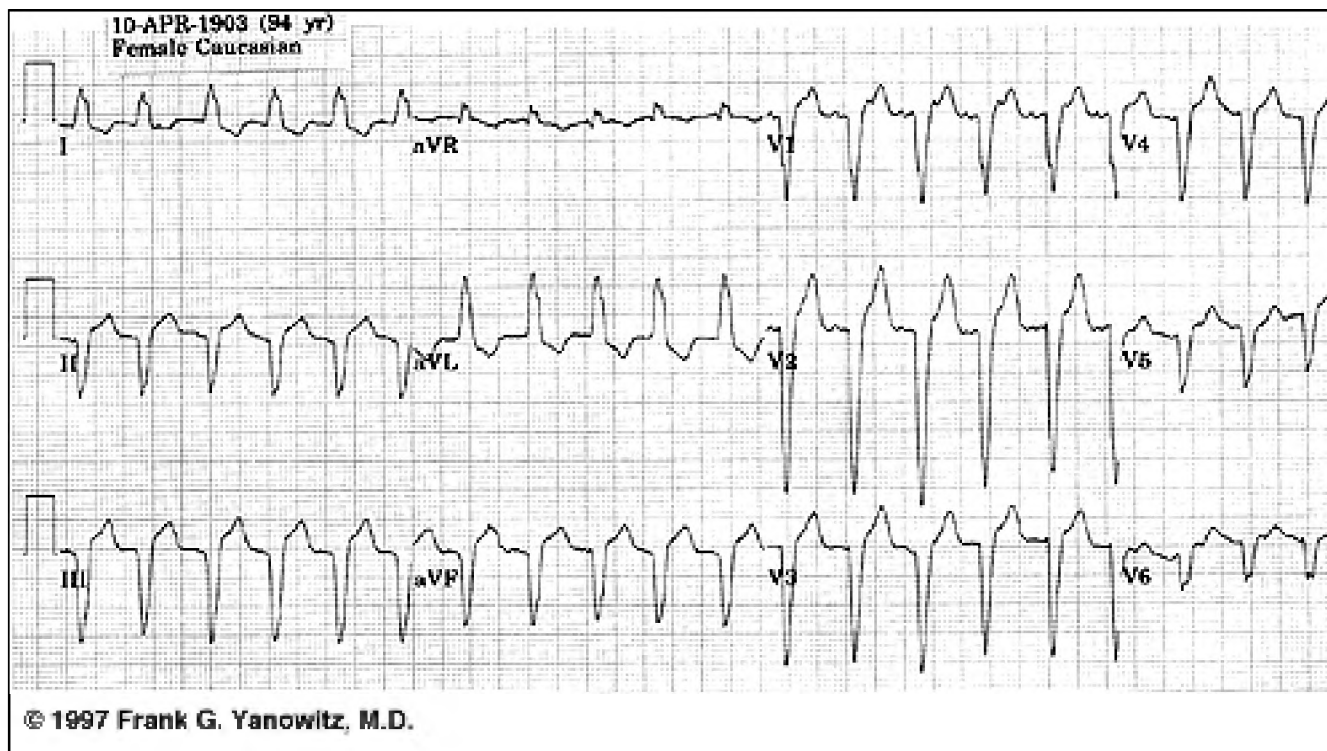
The basic rhythm is atrial flutter with variable AV block. When 2:1 conduction ratios occur there is a rate-dependent LBBB. Don't be fooled by the wide QRS tachycardia on the bottom strip. It's not ventricular tachycardia, but atrial flutter with 2:1 conduction and LBBB. Lidocaine is not needed because there is no ventricular ectopy.



## Atrial Flutter With 2:1 AV Conduction: Leads II, III, V1-KH

*Frank G. Yanowitz, M.D.*

In leads II and III, the one of the flutter waves occurs at the end of the QRS complex and might be mistaken for part of the QRS itself; i.e., the S wave. In lead V1, the two flutter waves for every QRS are more easily identified.

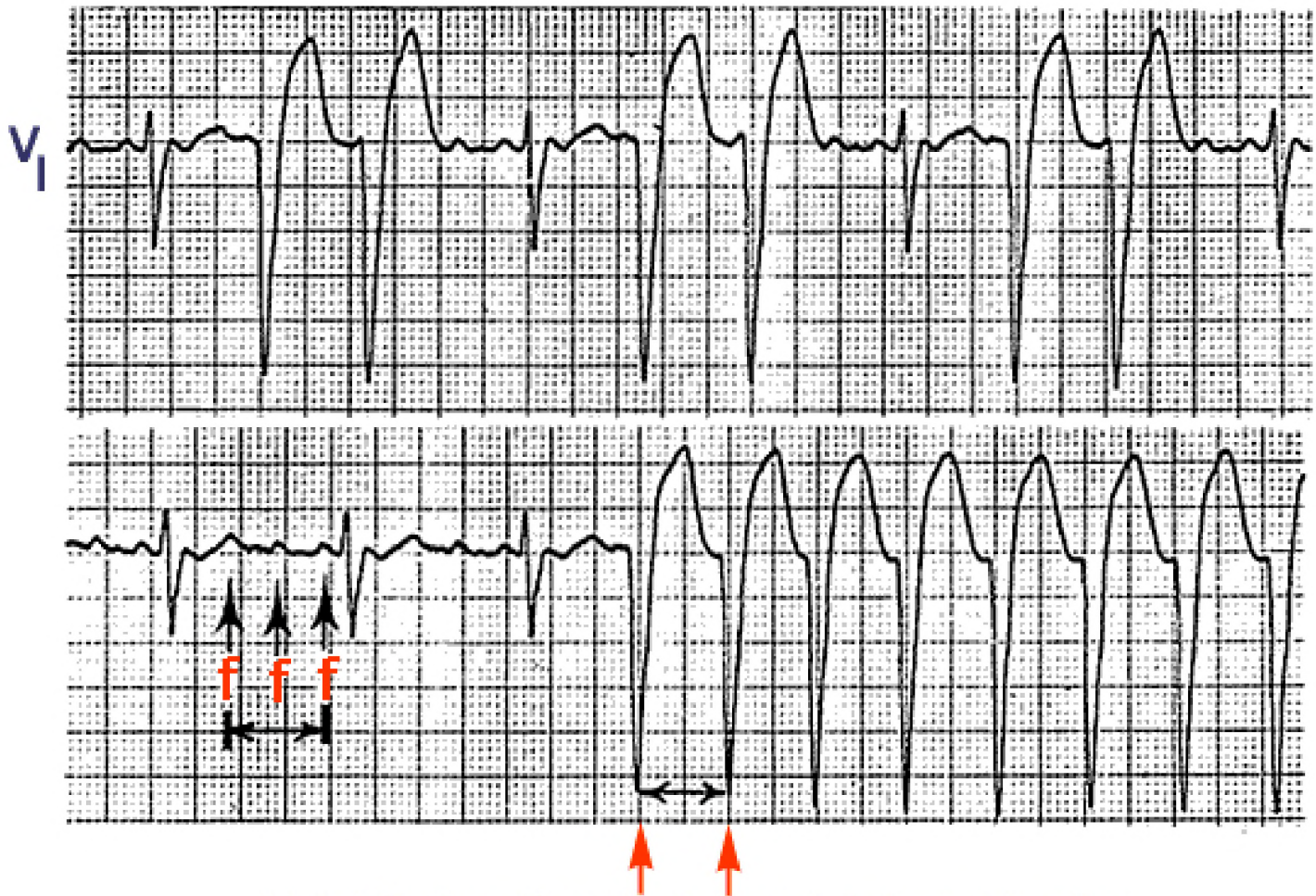


## LBBB and Atrial Flutter with 2:1 AV Block

*Frank G. Yanowitz, M.D. copyright 1997*

The LBBB is obvious by the monophasic R wave in leads I and aVL; the atrial flutter is less obvious, but in lead V1 atrial activity at 280/min can be seen in a 2:1 conduction pattern.



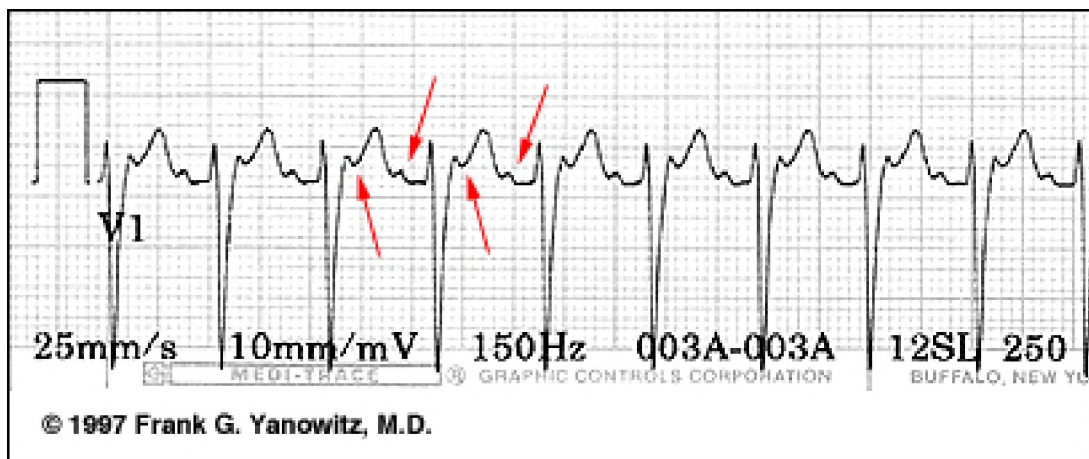


**Atrial flutter (f) with 2:1 and 4:1 conduction.  
Note rate-dependent LBBB**

## **Atrial Flutter With 2:1 and 4:1 Conduction and Rate Dependent LBBB-KH**

*Frank Yanowitz Copyright 1996*

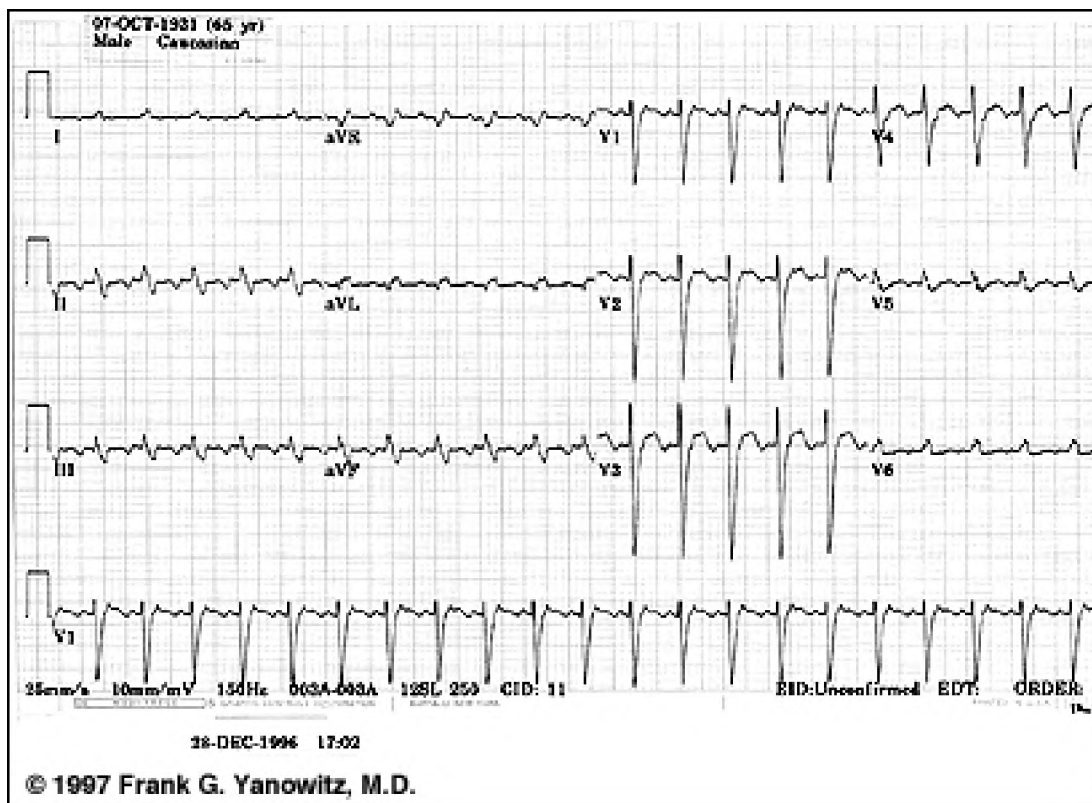
In this example of atrial flutter with variable AV conduction, the faster rates are associated with rate-related LBBB. Don't confuse this for ventricular tachycardia.



## Atrial Flutter With 2:1 AV Conduction: Lead V1-KH

*Frank G. Yanowitz, M.D.*

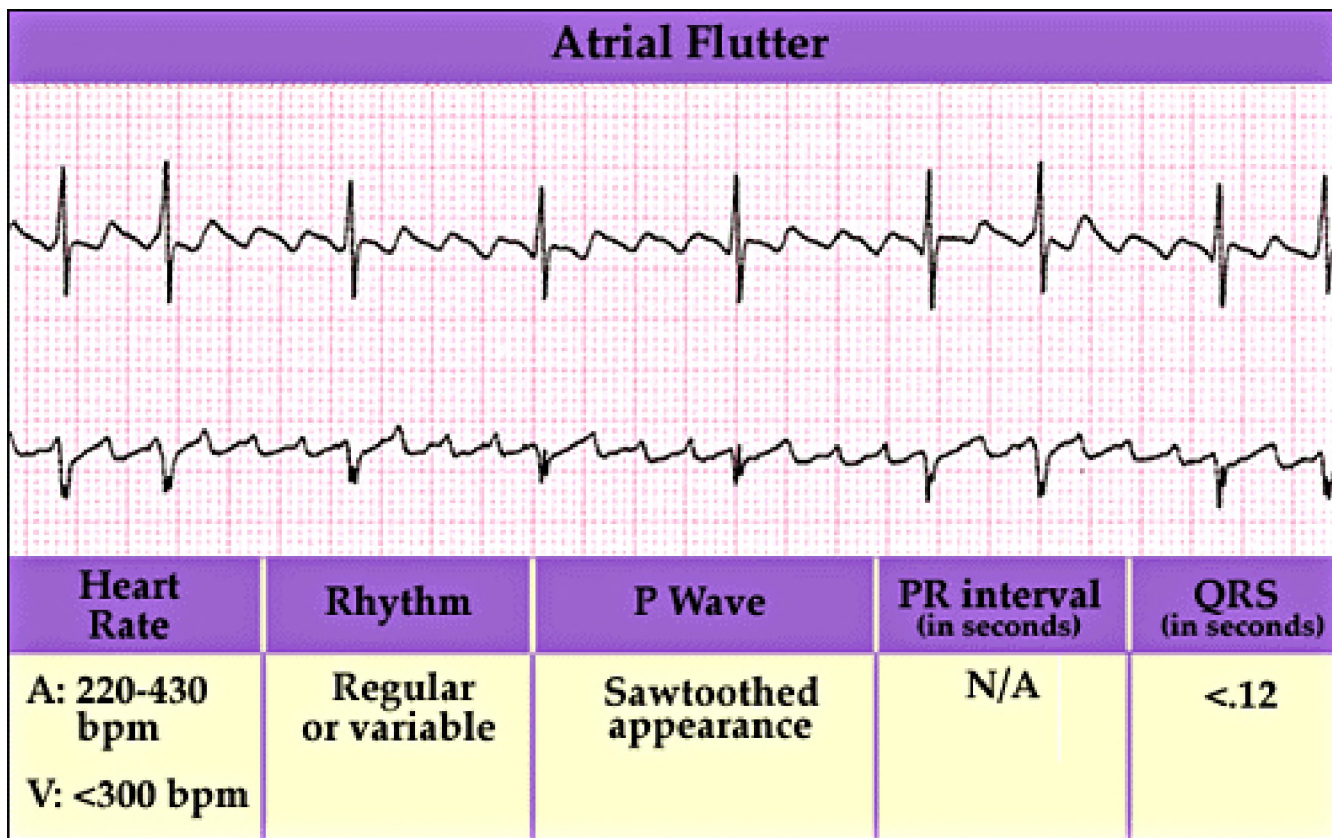
The arrows point to two flutter waves for each QRS complex. Atrial rate = 280; ventricular rate = 140.



## Atrial Flutter With 2:1 AV Conduction-KH

*Frank G. Yanowitz, M.D.*

Flutter waves are best seen in lead V1; one immediately follows the QRS and the other precedes the next QRS. The regular ventricular rate of 150 bpm should always prompt us to consider this diagnosis.



## Atrial Flutter With Variable AV Block - Marquette-KH

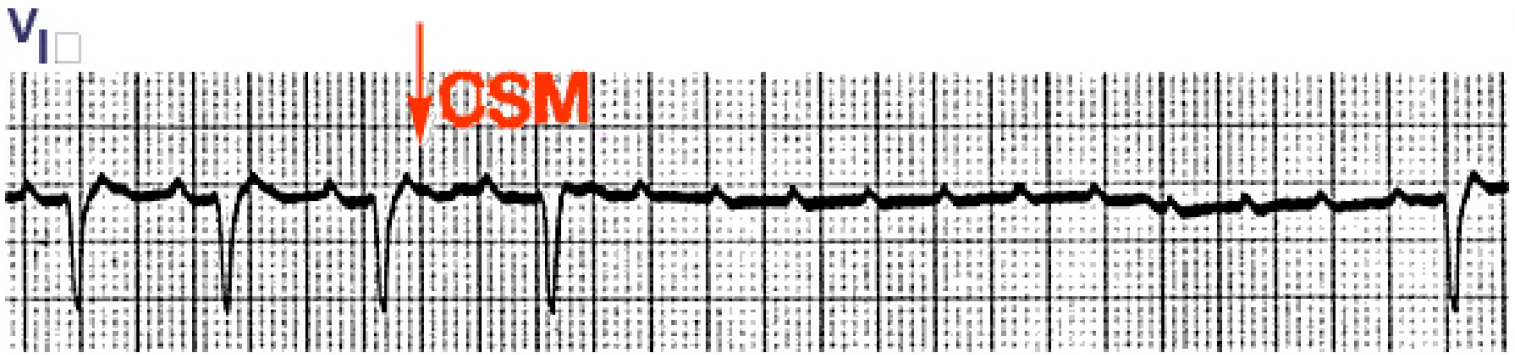
*Marquette Electronics Copyright 1996*





## Atrial Flutter With 2:1 Conduction: Leads II, III, V1-KH

*Frank G. Yanowitz, M.D.*



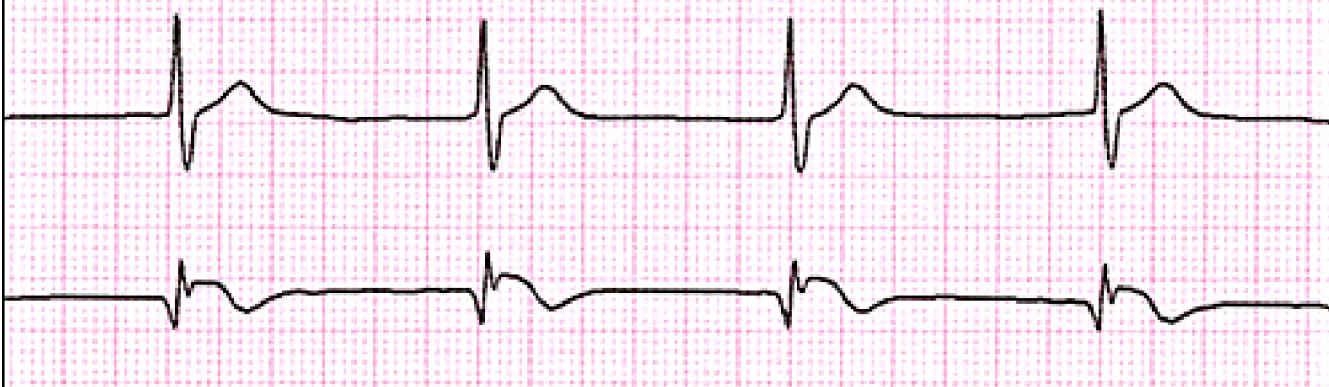
**Atrial flutter with 2:1 block responding to carotid sinus massage (CSM)**

## Massage Parlor Games-KH

*Frank Yanowitz Copyright 1996*

When unsure of the mechanism of a supraventricular tachycardia, carotid sinus massage may help make the diagnosis. In this example, carotid sinus massage causes marked AV block which permits easy recognition of the rapid, regular atrial flutter waves.

## Junctional Rhythm

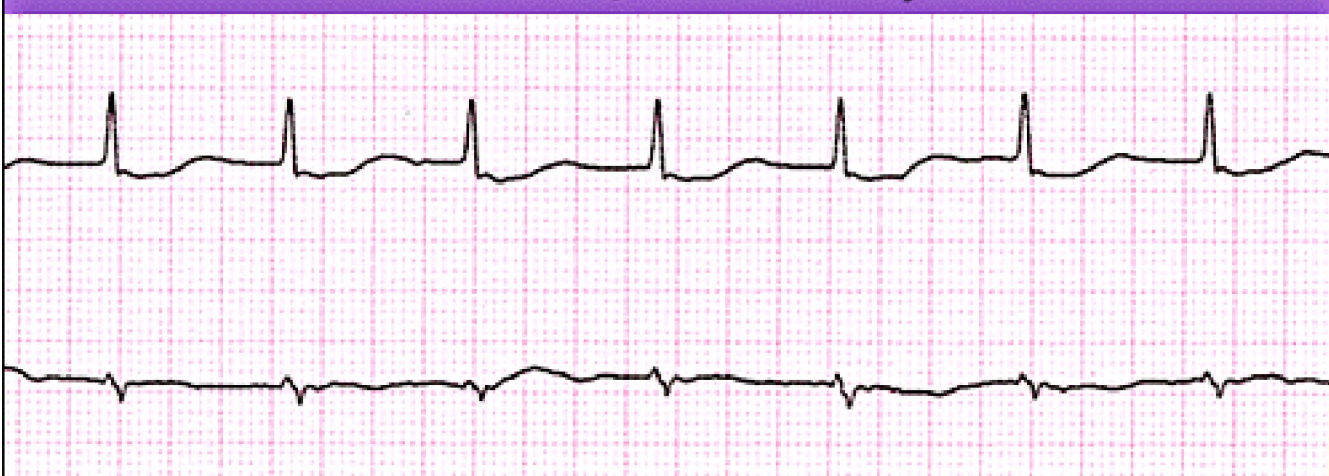


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
40-60 bpm	Regular	Inverted, absent or after QRS	<.12	<.12

## Junctional Escape Rhythm-KH

*Marquette Electronics Copyright 1996*

## Accelerated Junctional Rhythm



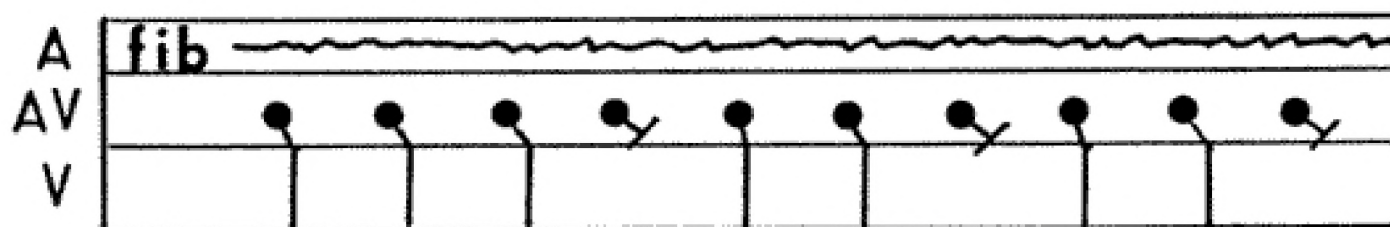
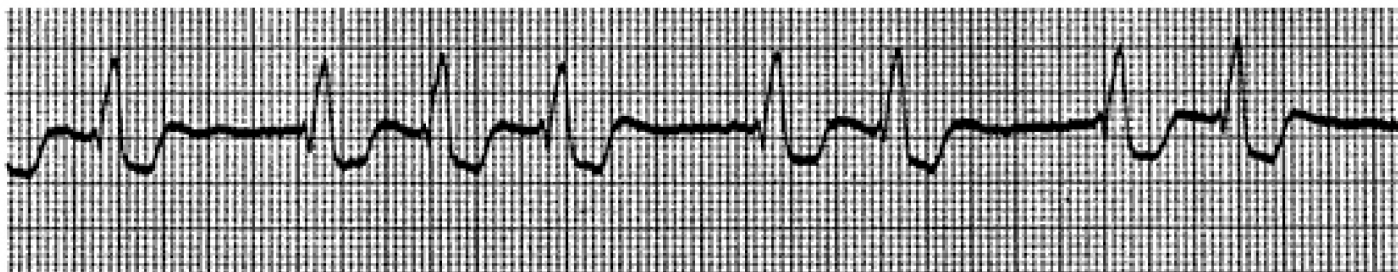
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
60-100 bpm	Regular	Inverted, absent or after QRS	<.12	<.12

## Accelerated Junctional Rhythm-KH

*Frank G. Yanowitz, M.D., copyright 1997*



## Lead V<sub>1</sub>



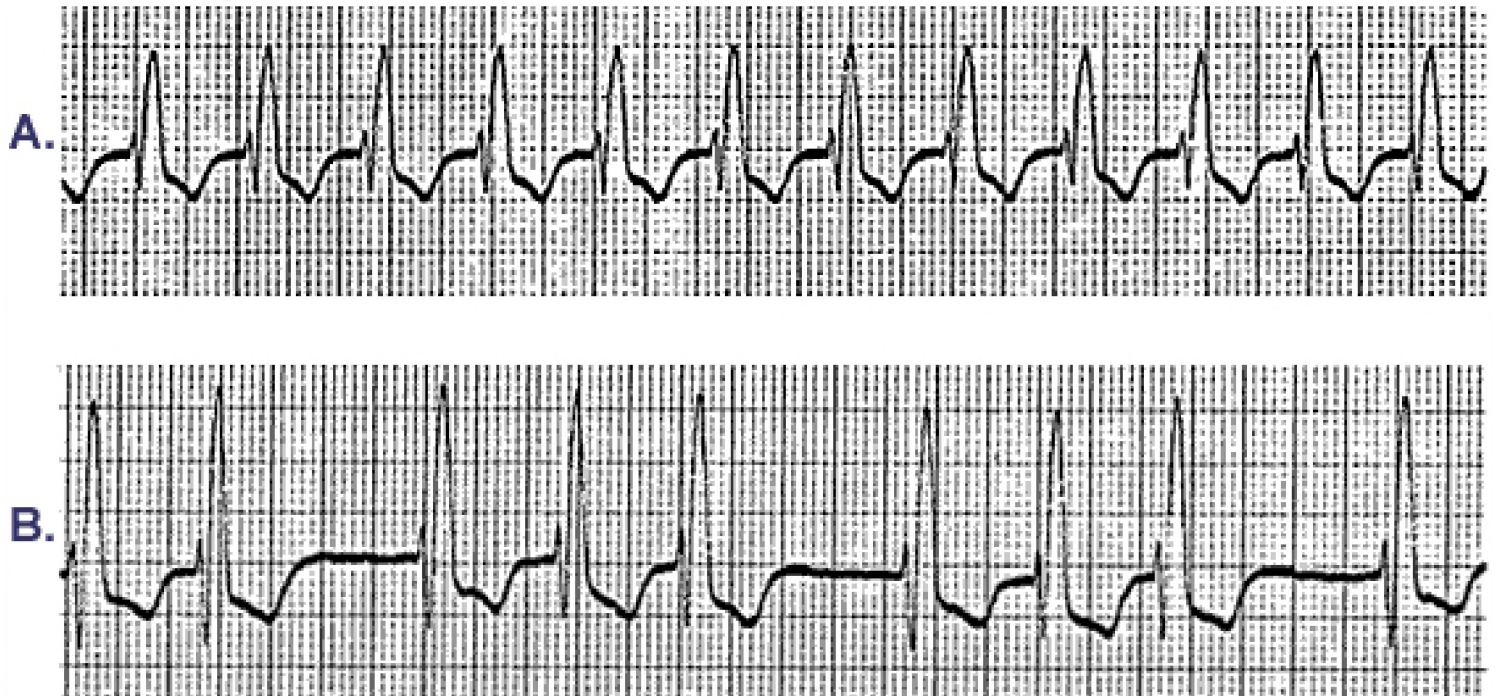
**Junctional tachycardia with exit block**

## Junctional Tachycardia With Exit Block: A Manifestation of Digitalis Intoxication-KH

*Frank Yanowitz Copyright 1996*

The "ladder diagram" says it all: the atria are fibrillating; there is complete heart block in the AV junction; a junctional tachycardia focus is firing at about 130 bpm, but not all junctional impulses reach the ventricles due to 2nd degree exit block.

**History: 76yo; longstanding atrial fib and RBBB**  
**Lead V<sub>1</sub>**  
**Treated with Digoxin and diuretics**



**A. What's the rhythm?**

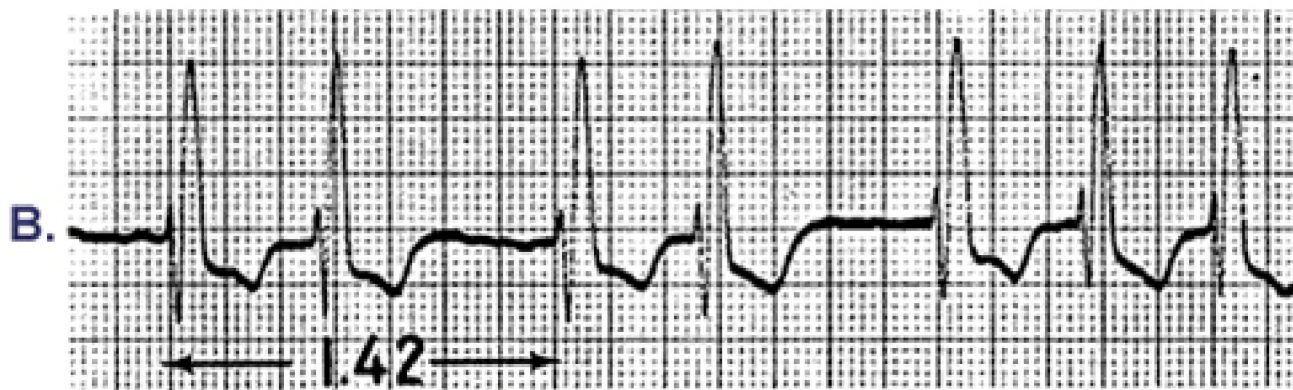
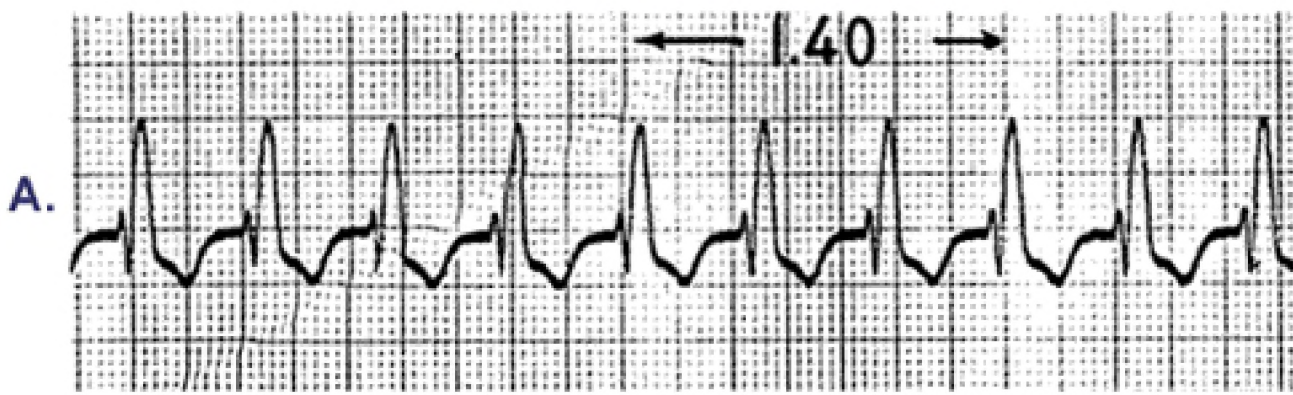
**B. (6 hrs later) What's the rhythm?**

## **Digitalis Intoxication: Junctional Tachycardia With and Without AV Block-KH**

*Frank Yanowitz Copyright 1996*

In a patient with longstanding atrial fibrillation being treated with digoxin, a regular tachycardia, as seen in 'A', with a RBBB suggests a junctional or supraventricular tachycardia. Group beating, in 'B', is likely due to a 2nd degree, Type 1, exit block below the ectopic junctional focus. This is highly suggestive of digitalis intoxication.

## Diagnosis: Digitalis Intoxication



A. Junctional tachycardia

B. Junctional tachycardia with exit block

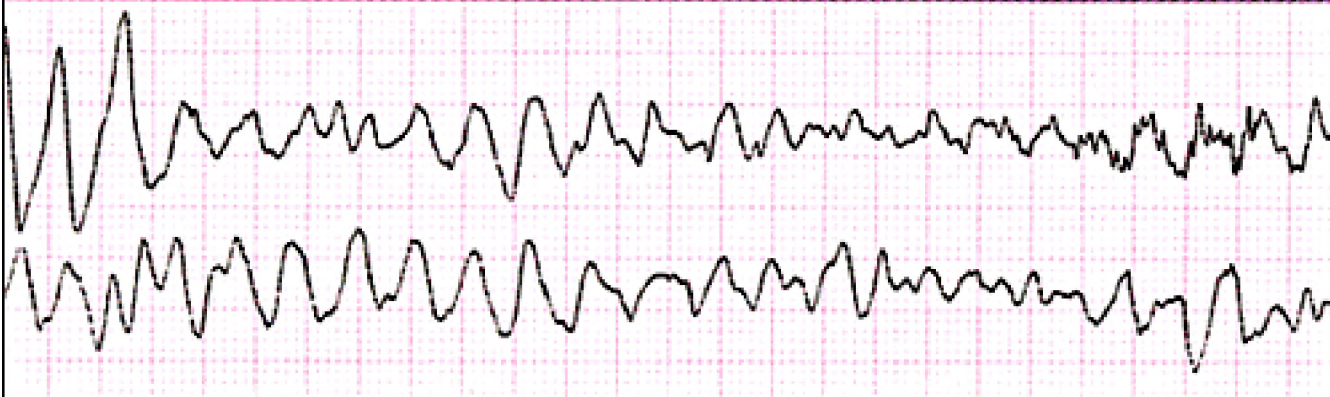
## Digitalis Intoxication: Junctional Tachycardia With and Without Exit Block-KH

*Frank Yanowitz Copyright 1996*

In 'A' the rhythm is junctional tachycardia with RBBB. In 'B' there is 2nd degree exit block with a 3:2 conduction ratio; i.e., every 3rd junctional impulse fails to reach the ventricles... at least for the first two groupings on 1.4sec.



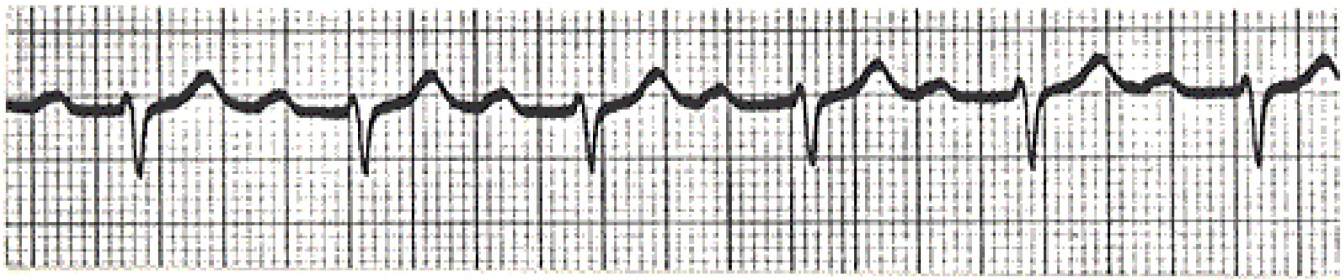
## Ventricular Fibrillation



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
300-600	Extremely irregular	Absent	N/A	Fibrillatory baseline

## Ventricular Fibrillation - Marquette-KH

*Marquette Electronics Copyright 1996*

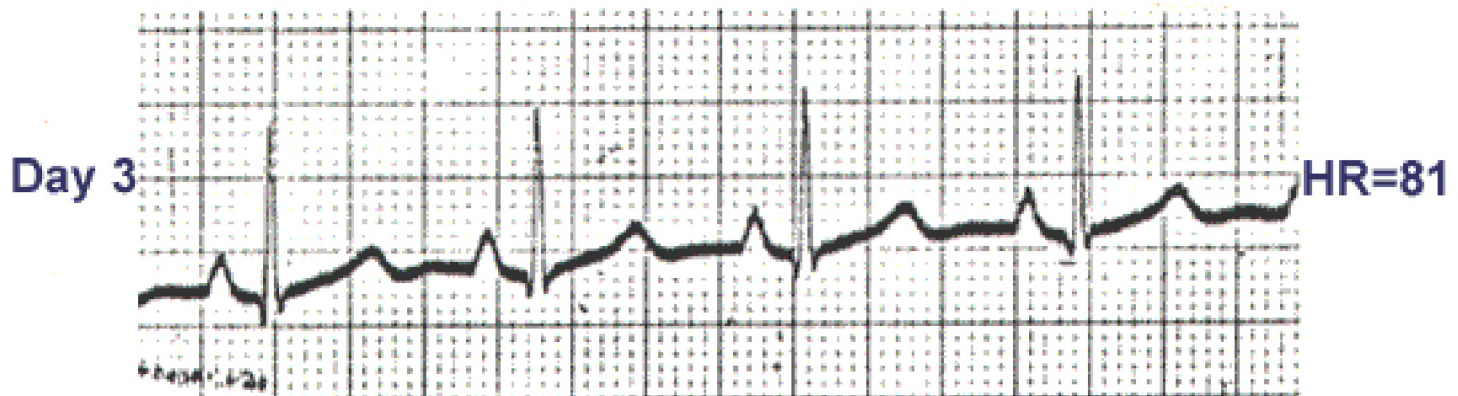
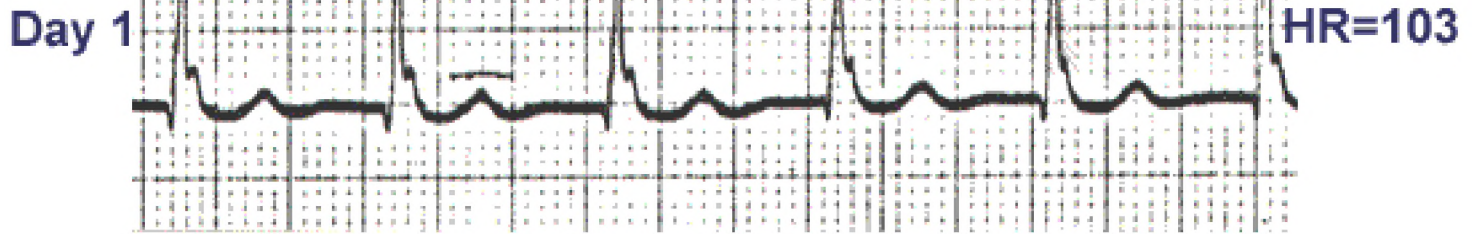


**1st degree AV block (PR = 280 ms)**

## 1st Degree AV Block

*Frank Yanowitz Copyright 1996*

The normal PR interval is 0.12 - 0.20 sec, or 120 -to- 200 ms. 1st degree AV block is defined by PR intervals greater than 200 ms. This may be caused by drugs, such as digoxin; excessive vagal tone; ischemia; or intrinsic disease in the AV junction or bundle branch system.

**Lead II****Mischief in the AV node**

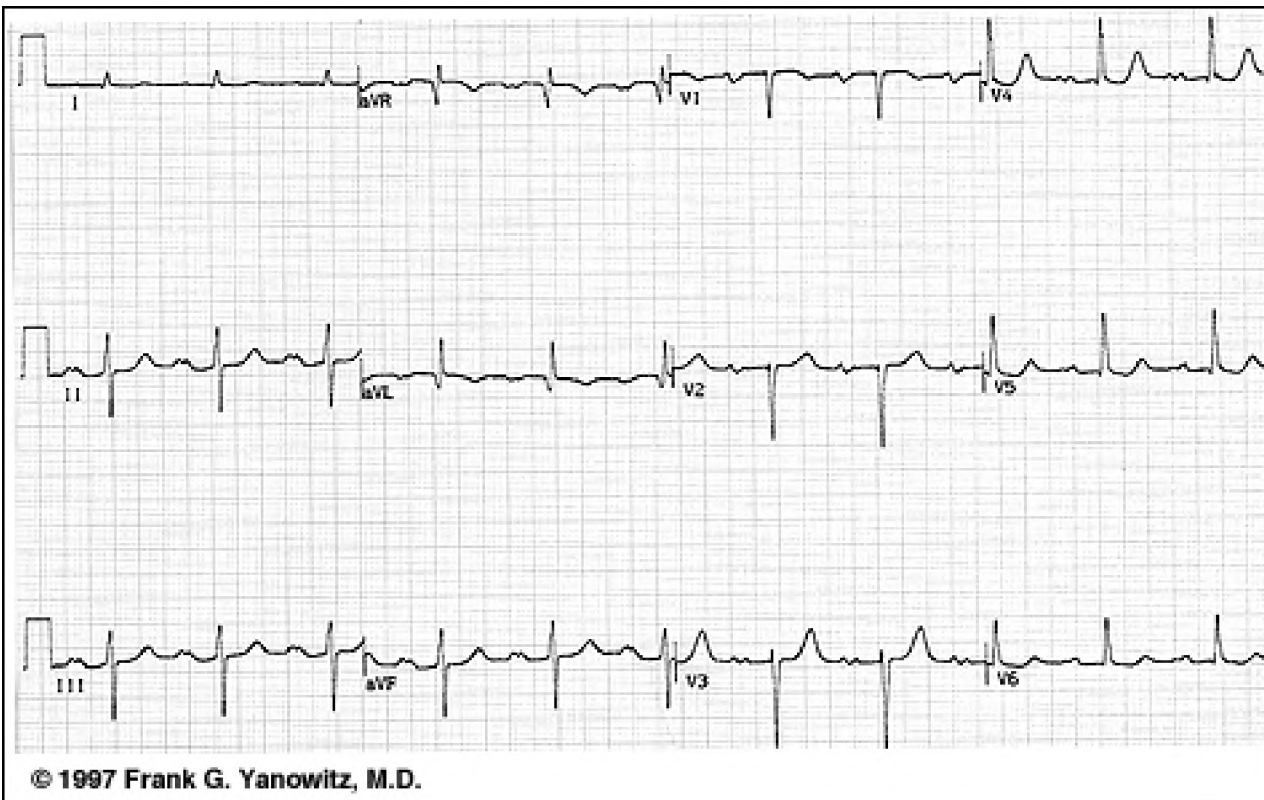
# ECG Of The Century: A Most Unusual 1st Degree AV Block

*Frank Yanowitz Copyright 1996*

On Day 1, at a heart rate of 103 bpm the P waves are not clearly defined suggesting an accelerated junctional rhythm. However, on Day 2, at a slightly slower heart rate the sinus P wave suddenly appears immediately after the QRS complex. In retrospect, the sinus P wave in Day 1 was found burried in the preceding QRS; note the notch on the downstroke of the QRS. On Day 3 a normal PR interval was seen.

How long can the PR interval get in 1st degree AV block??? No one knows.





## Left Atrial Abnormality & 1st degree AV Block-KH

*Frank G. Yanowitz, M.D.*

The P-wave is notched, wider than 0.12s, and has a prominent negative (posterior) component in V1 - all criteria for left atrial abnormality or enlargement (LAE). The PR interval  $>0.20$ s. Minor ST-T wave abnormalities are also present.



## Lead V<sub>1</sub>



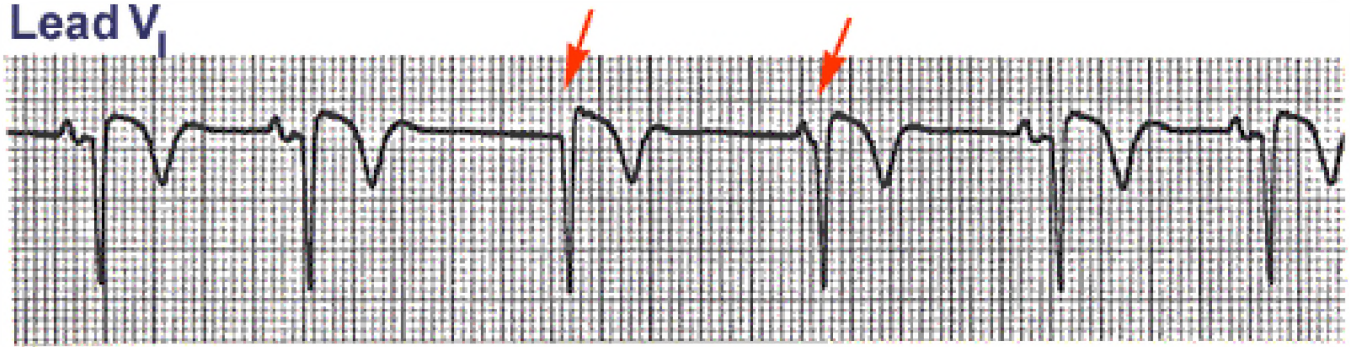
**What kind of AV heart block?**

## A Very Subtle 1st Degree AV Block

*Frank Yanowitz Copyright 1996*

Where are the P waves??? They are hiding in the T waves as indicated by the arrows. How do we know? The PVC unmasks the sinus P wave, and now it is seen in the pause following the PVC. The PR interval is, therefore, about 500 ms.

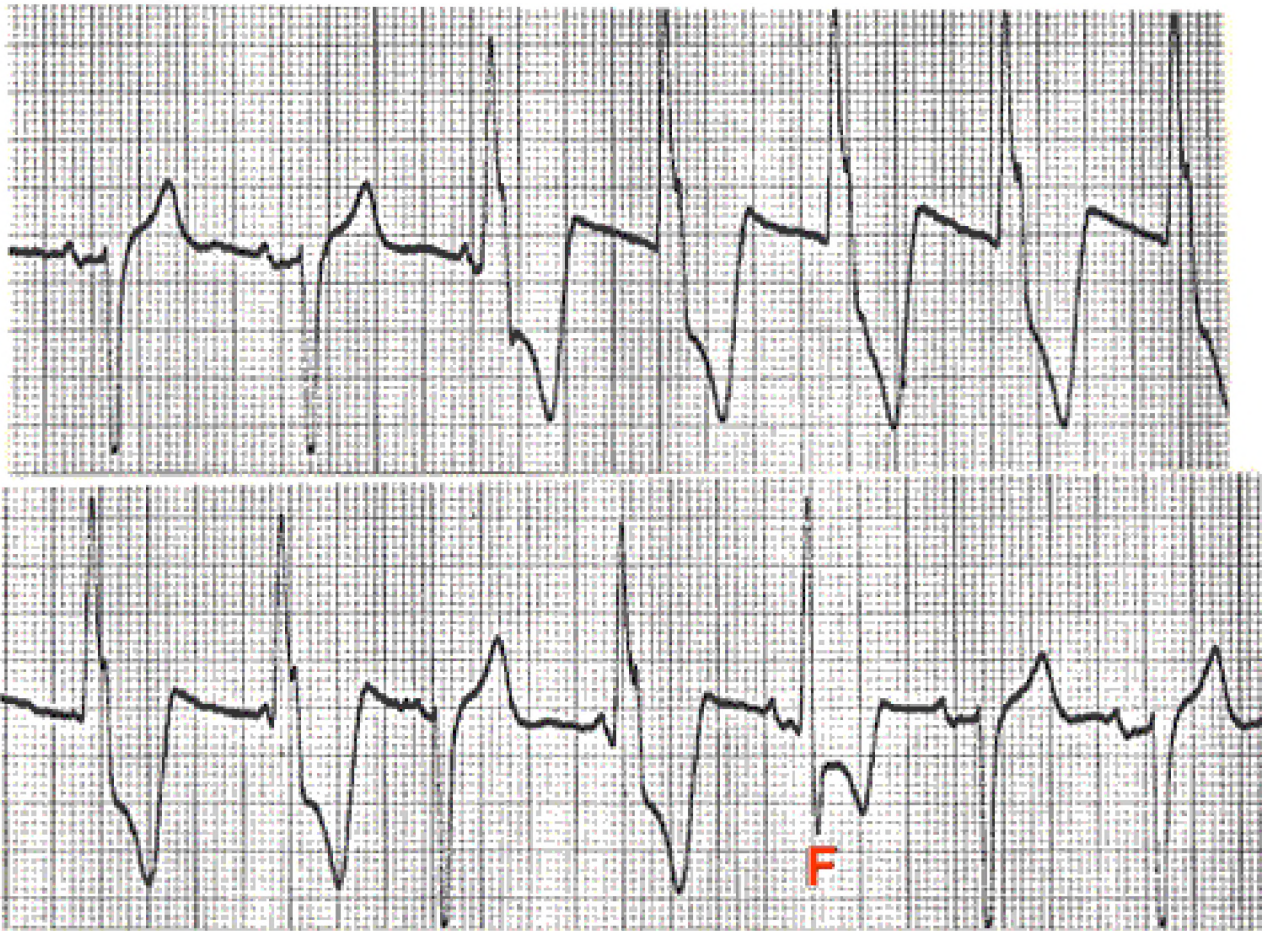
**Lead V<sub>1</sub>**



**Incomplete AV dissociation due to sinus slowing (default)  
with junctional escapes (arrows)**

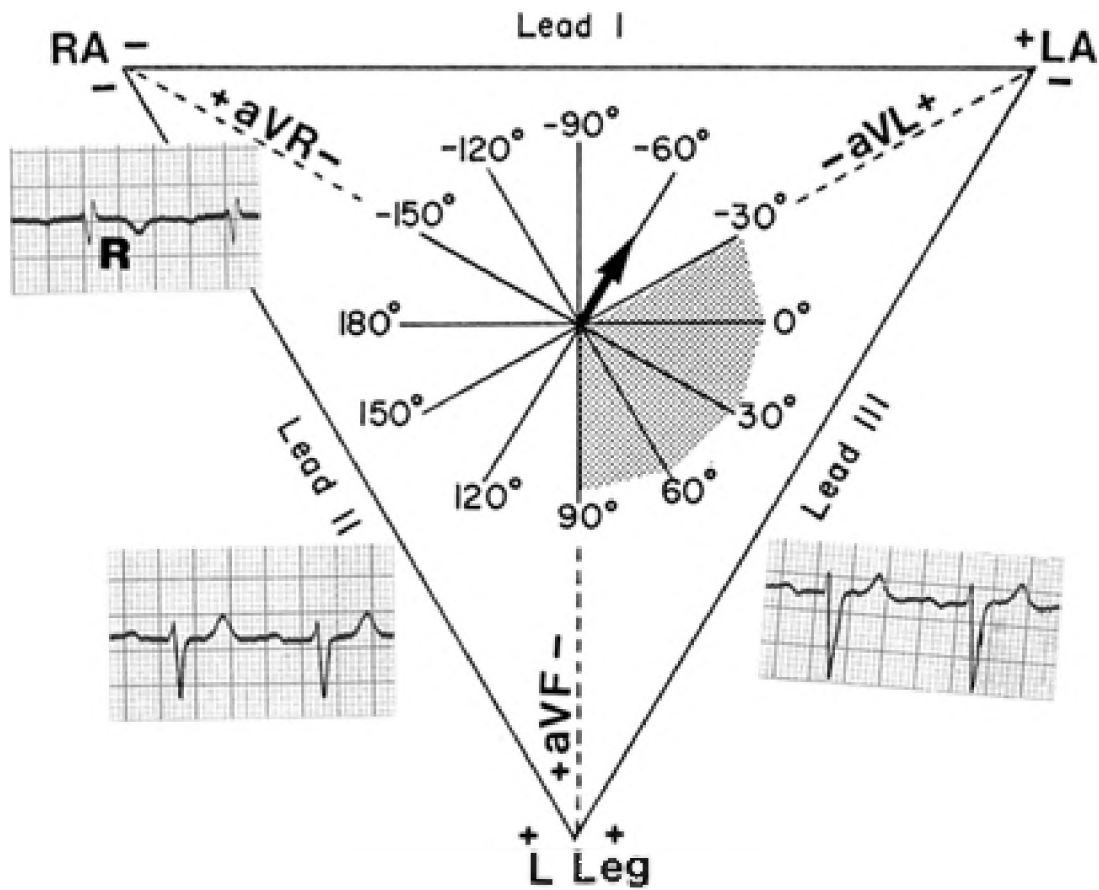


## Lead V<sub>1</sub>

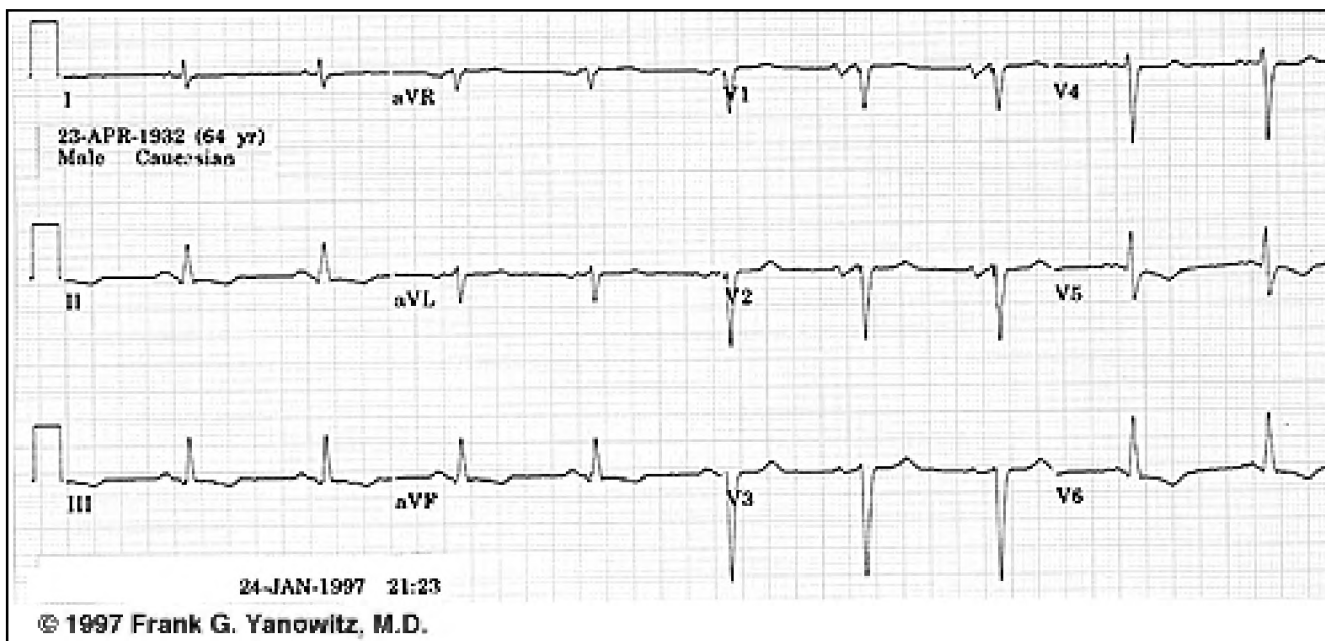


**Incomplete AV dissociation (usurpation)  
due to accelerated ventricular rhythm**

**F = fusion beat**



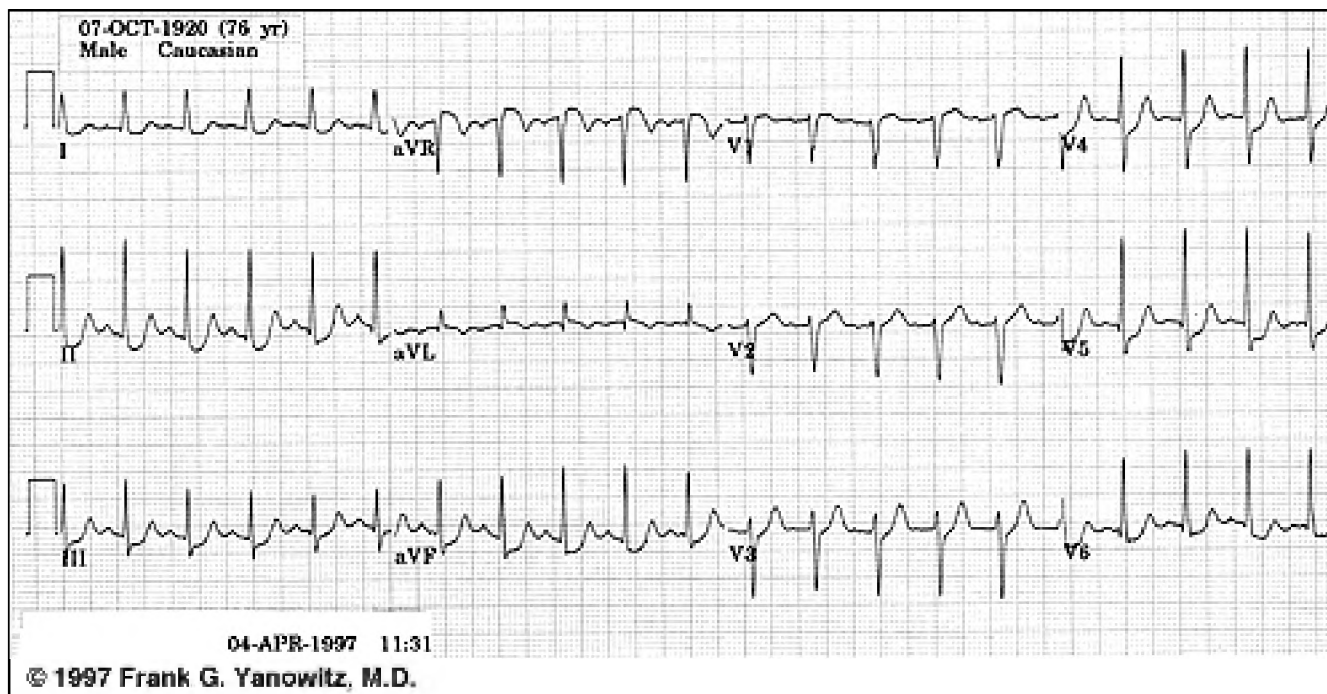
- Lead aVR is the smallest and isoelectric lead.
- The two perpendiculars are  $-60^\circ$  and  $+120^\circ$ .
- Leads II and III are mostly negative (i.e., moving away from the + left leg)
- The axis, therefore, is  $-60^\circ$ .



## Left Atrial Enlargement & Nonspecific ST-T Wave Abnormalities-KH

*Frank G. Yanowitz, M.D.*

LAE is best seen in V1 with a prominent negative (posterior) component measuring 1mm wide and 1mm deep. There are also diffuse nonspecific ST-T wave abnormalities which must be correlated with the patient's clinical status. Poor R wave progression in leads V1-V3, another nonspecific finding, is also present.

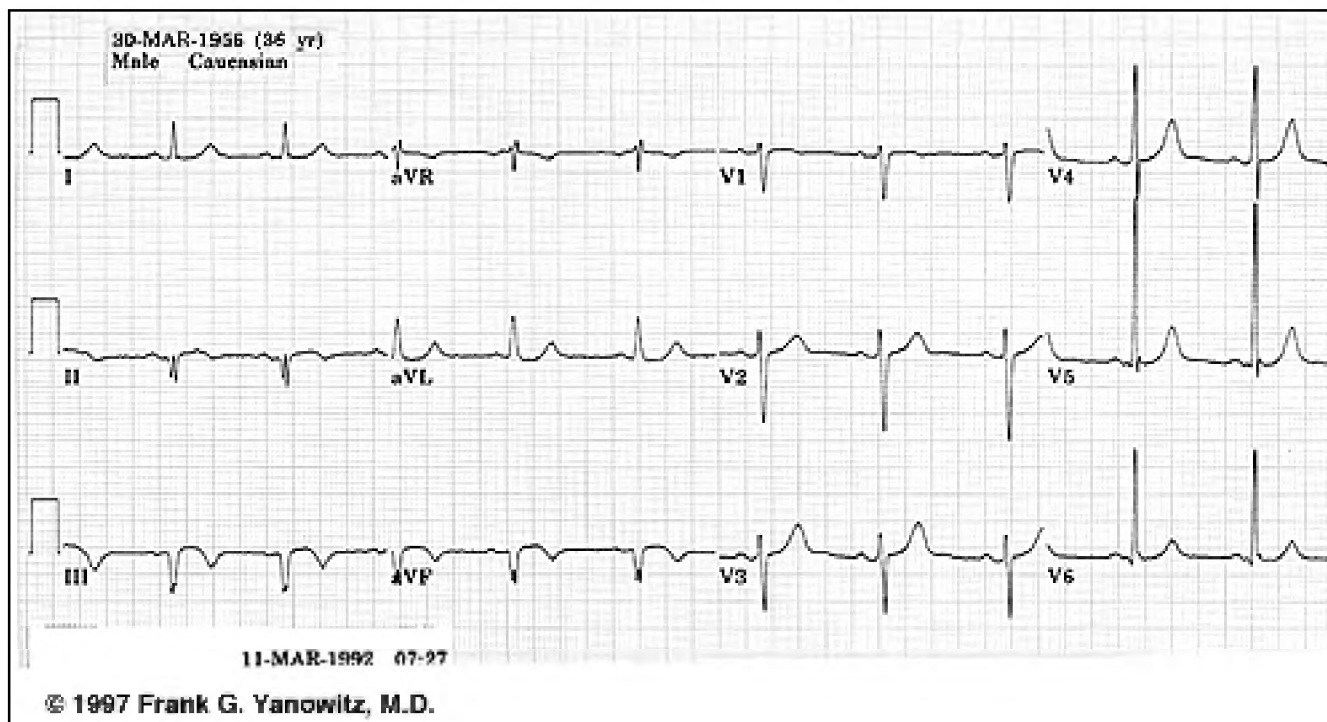


## ST Segment Depression-KH

*Frank G. Yanowitz, M.D.*

ST segment depression is a nonspecific abnormality that must be evaluated in the clinical context in which it occurs. In a patient with angina pectoris ST depression usually means subendocardial ischemia and, unlike ST elevation, is not localizing to a particular coronary artery lesion.

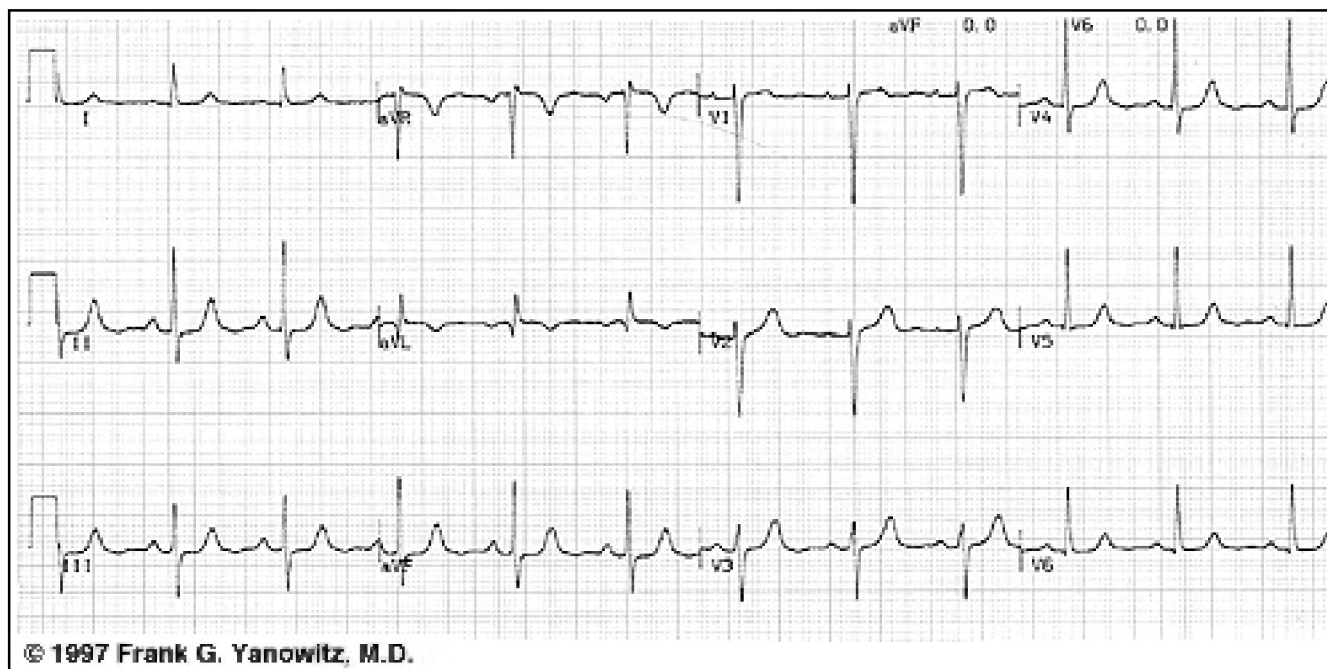




## Inferior MI: Fully Evolved-KH

*Frank G. Yanowitz, M.D.*

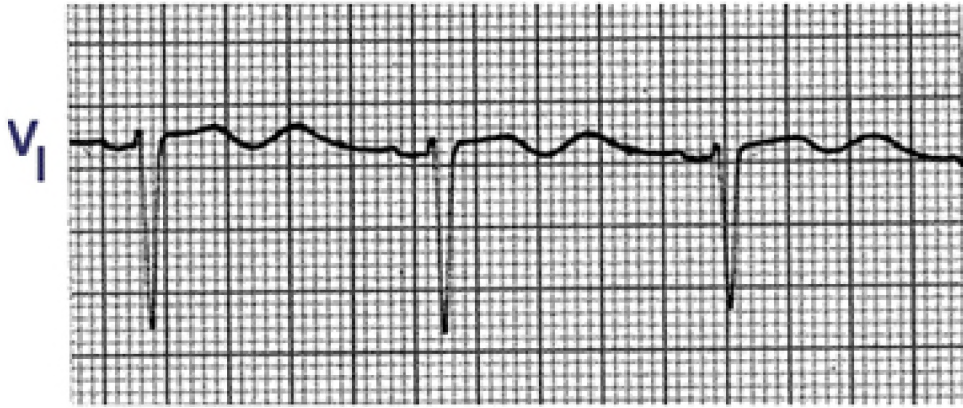
Significant pathologic Q-waves are seen in leads II, III, aVF along with resolving ST segment elevation and symmetrical T wave inversion. This is a classic inferior MI.



## High Lateral Wall MI (seen in aVL)-KH

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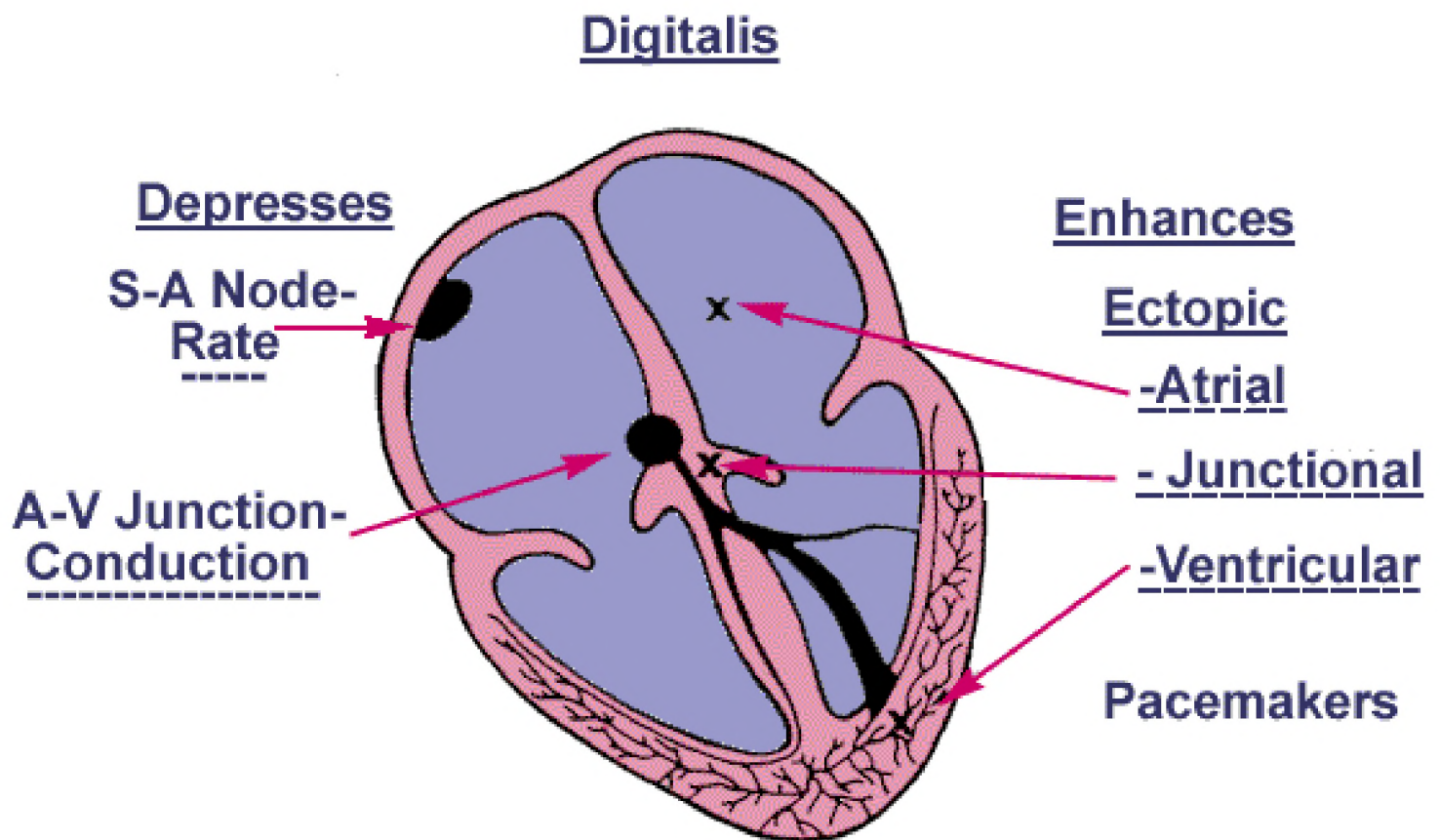


'U'-nbelievable

## Giant TU Fusion Waves-KH

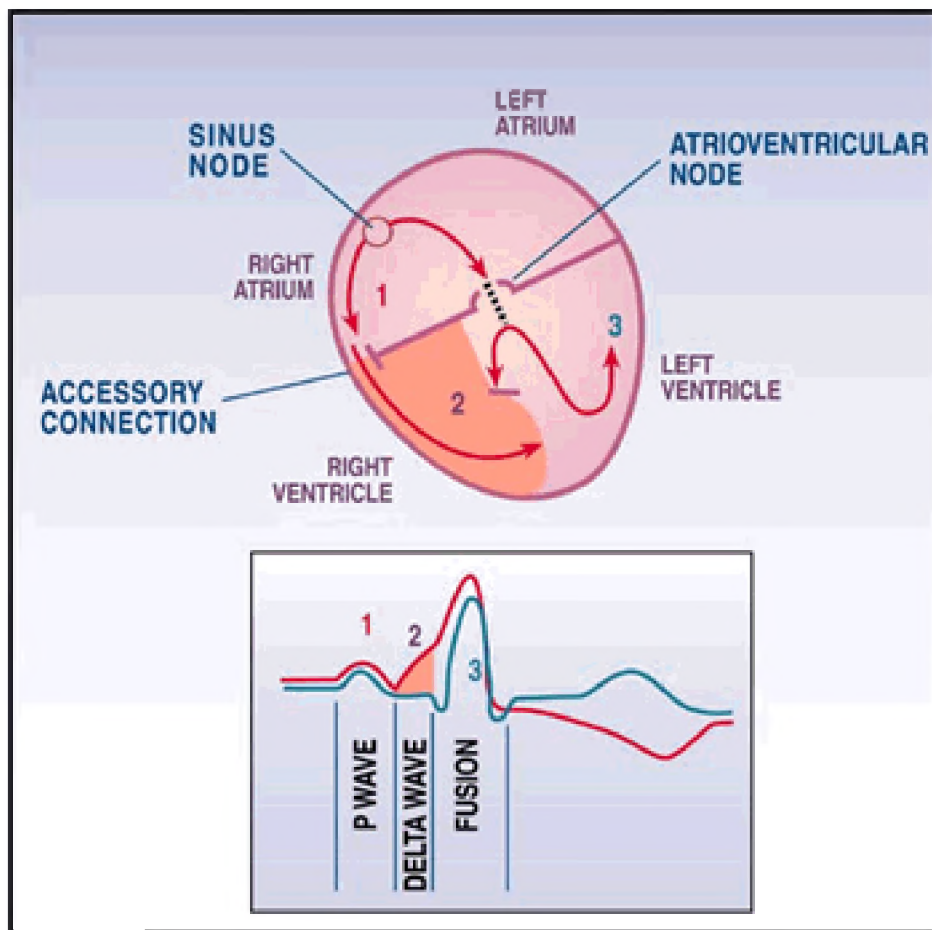
*Frank Yanowitz Copyright 1996*

TU fusion waves are often seen in long QT syndromes. The differential diagnosis of this ECG abnormality includes electrolyte abnormalities -hypokalemia, CNS disease, e.g., subarachnoid hemorrhage; hereditary long QT syndromes, and drugs such as quinidine.



## Diagram: Digitalis Effect on Rhythm and Conduction-KH

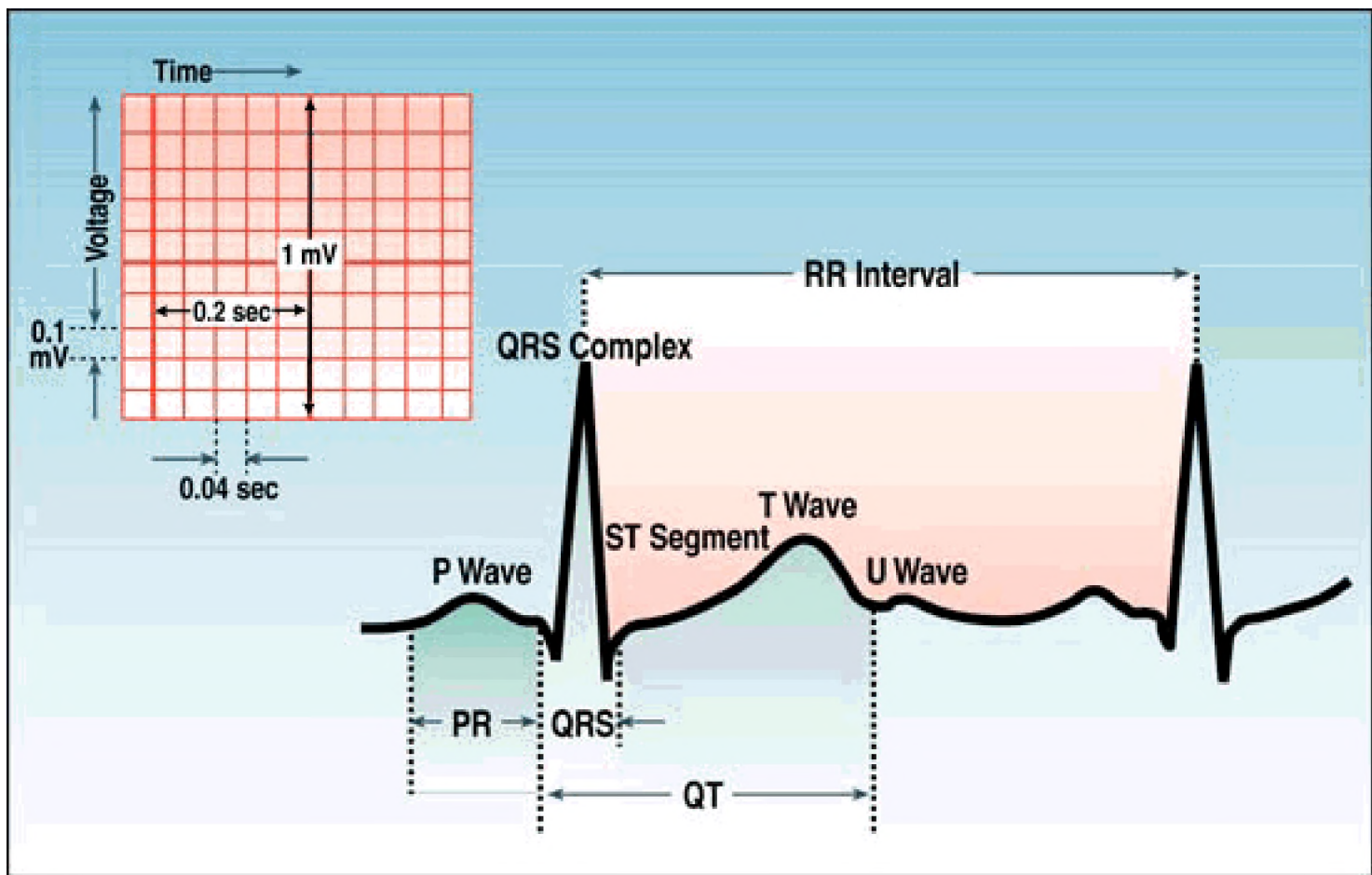
*Frank Yanowitz Copyright 1996*



## WPW Diagram-KH

*Frank G. Yanowitz, M.D., copyright 1997*

The short PR interval is due to a bypass track, also known as the Kent pathway. By bypassing the AV node the PR shortens. The delta wave represents early activation of the ventricles from the bypass tract. The fusion QRS is the result of two activation sequences, one from the bypass tract and one from the AV node. The ST-T changes are secondary to changes in the ventricular activation sequence.

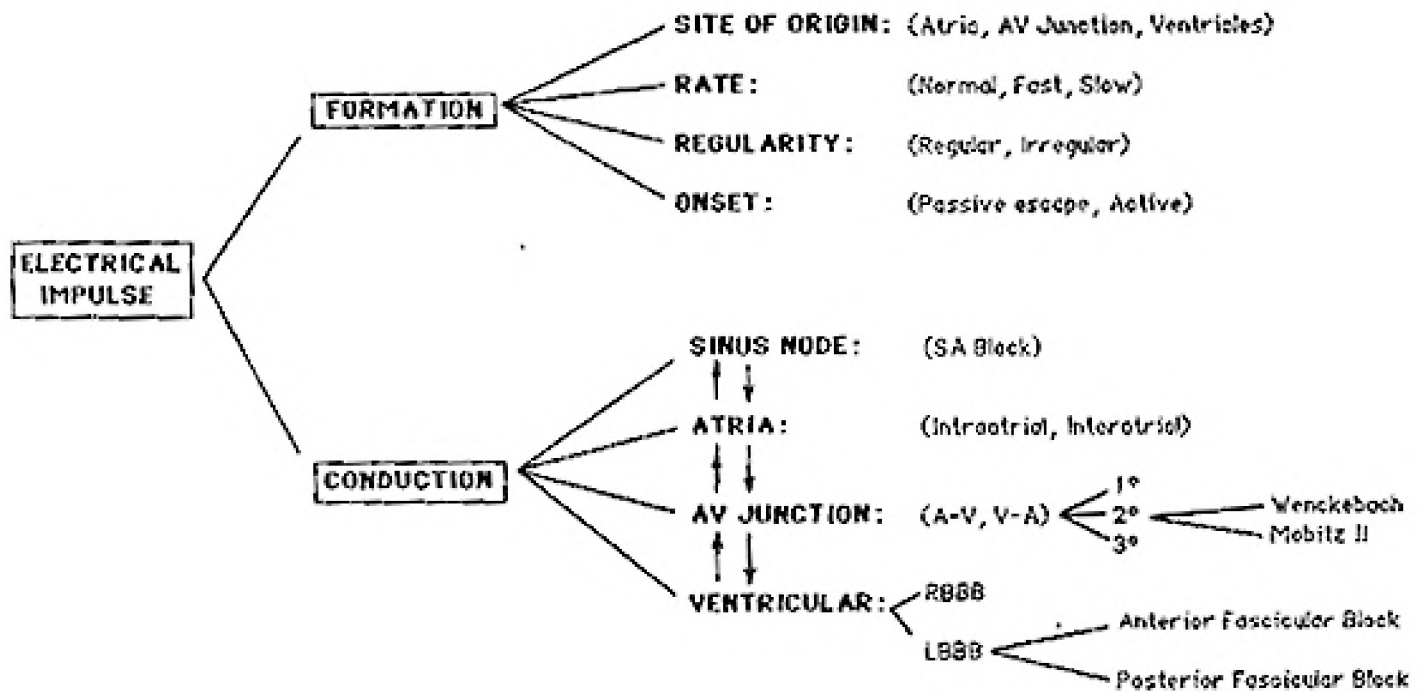


## ECG Intervals and Waves-KH

*Frank G. Yanowitz, M.D., copyright 1997*

The P wave represents atrial activation; the PR interval is the time from onset of atrial activation to onset of ventricular activation. The QRS complex represents ventricular activation; the QRS duration is the duration of ventricular activation. The ST-T wave represents ventricular repolarization. The QT interval is the duration of ventricular activation and recovery. The U wave probably represents "afterdepolarizations" in the ventricles.

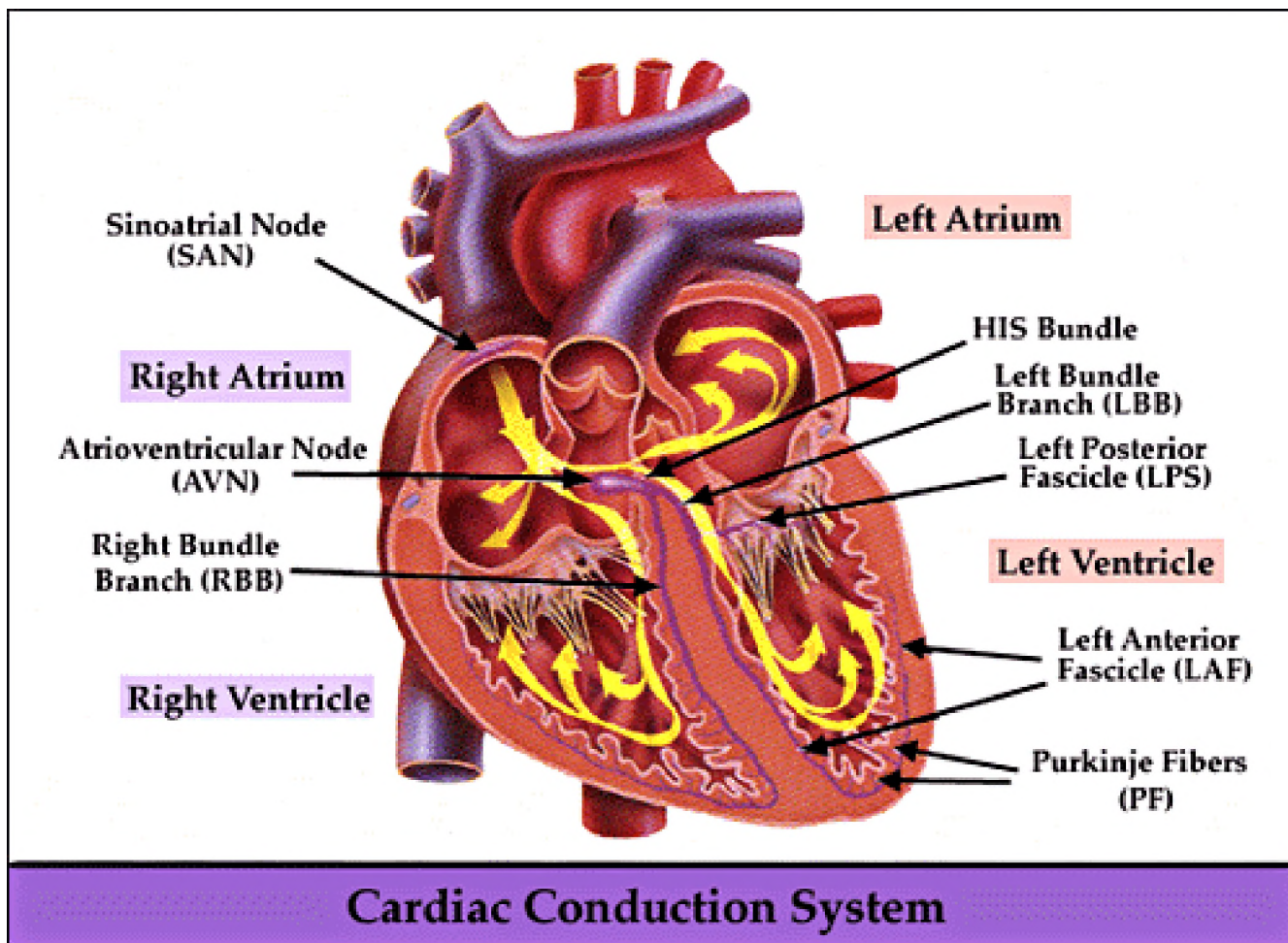
## HOW TO THINK ABOUT ARRHYTHMIAS AND CONDUCTION DISTURBANCES



## Conceptual Framework: Arrhythmias and Conduction Abnormalities-KH

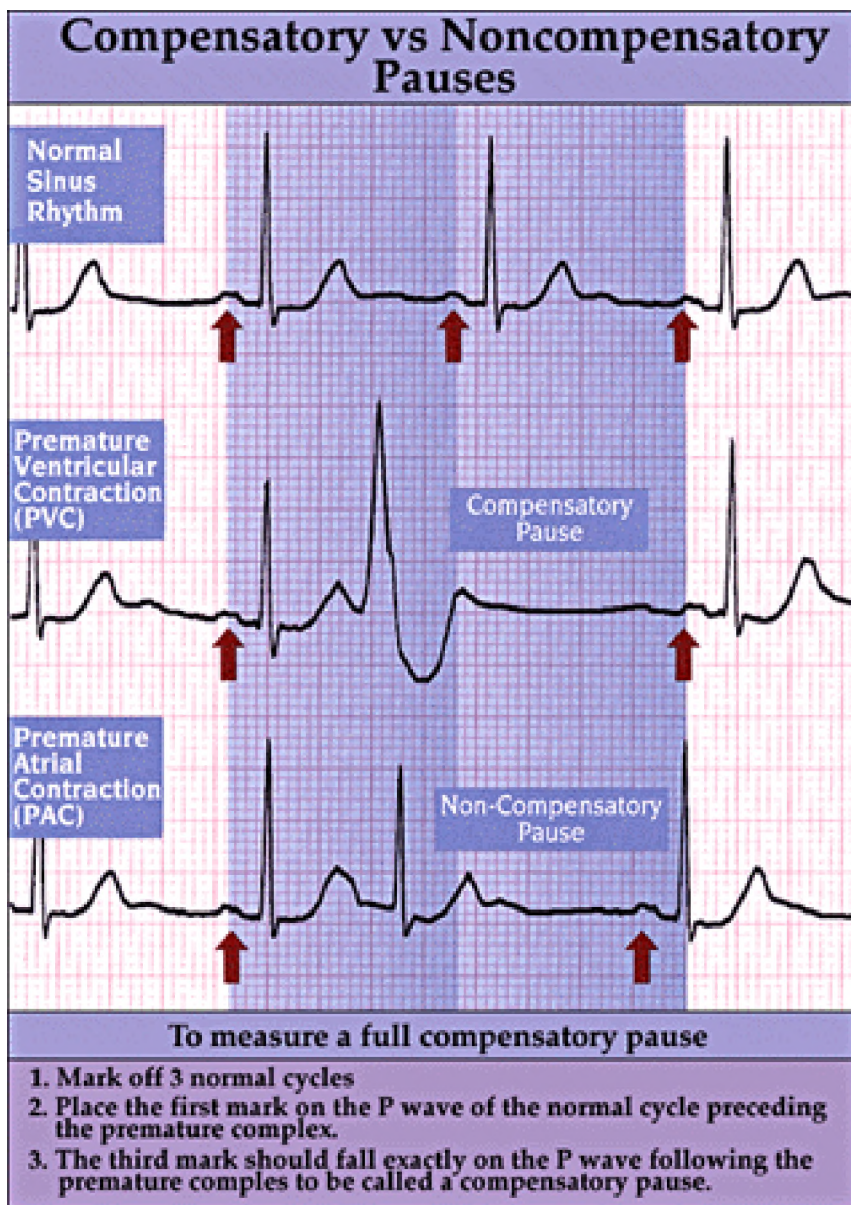
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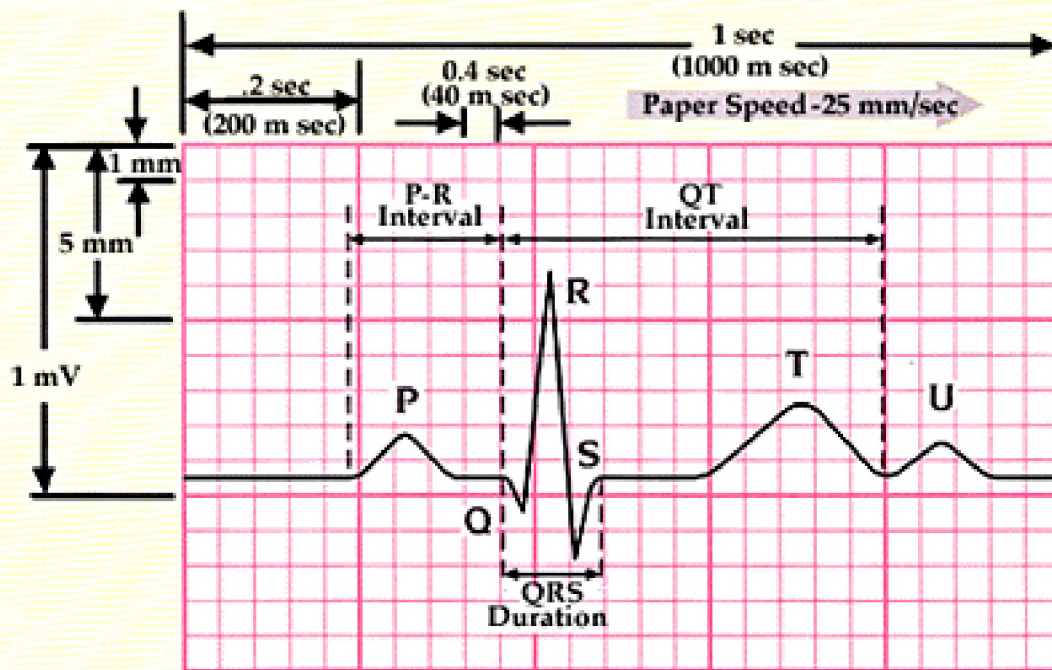
# Cardiac Conduction System Diagram - Marquette-KH

*Marquette Electronics Copyright 1996*



## Compensatory vs. Non-compensatory Pauses - Marquette-KH

*Marquette Electronics Copyright 1996*



**Verticle Axis**

1 small square = 1mm (0.1mV)  
 1 Large Square = 5mm (0.5mV)  
 2 large squares = 1mV

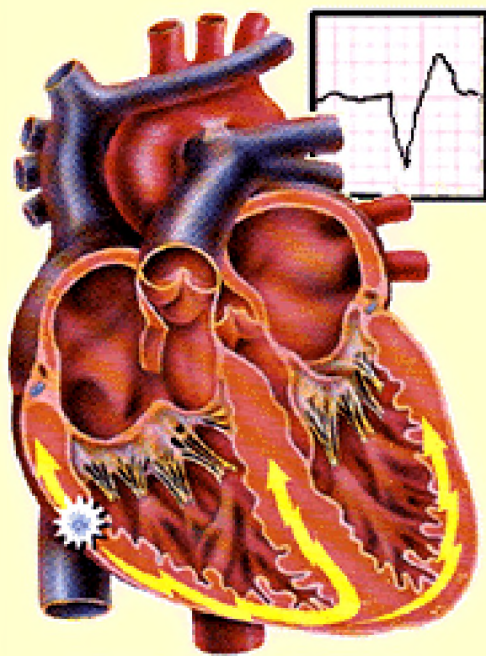
**Horizontal Axis**

1 small square = .04 (40 m sec)  
 1 large square = .2 (200 m sec)  
 5 large squares = 1 sec (1000 m sec)

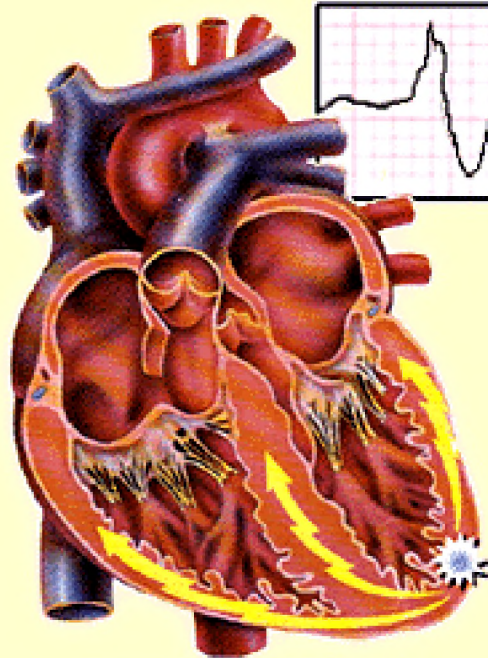
## ECG Components Diagram - Marquette-KH

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Right Ventricular PVC

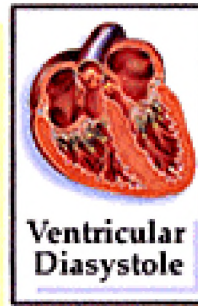
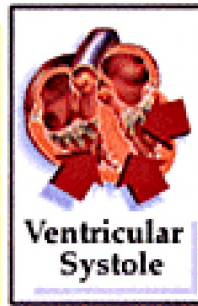
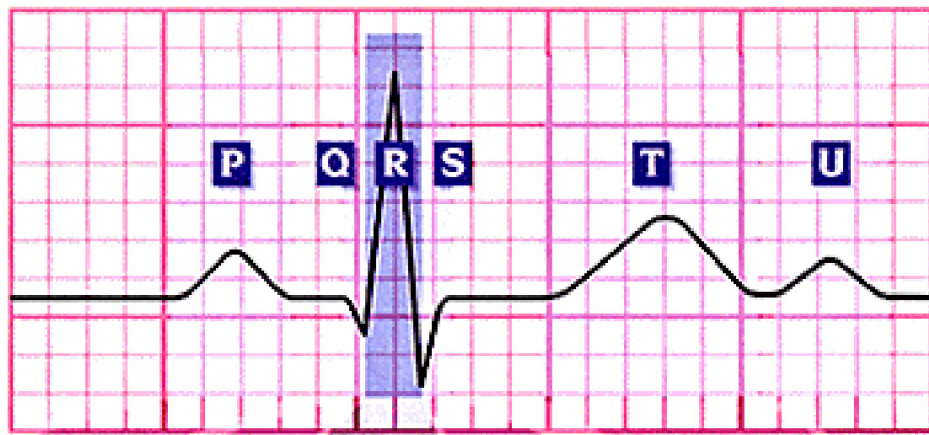


Left Ventricular PVC

## Ventricular Conduction

# RV vs LV PVC's - Marquette-KH

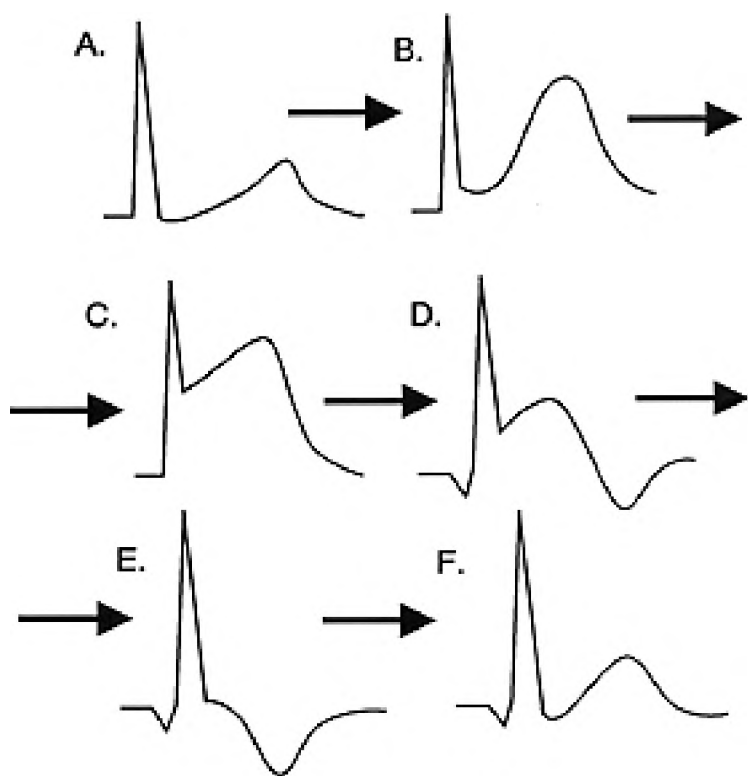
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## Electrical and Mechanical Events

# Electrical and Mechanical Events Diagram - Marquette-KH

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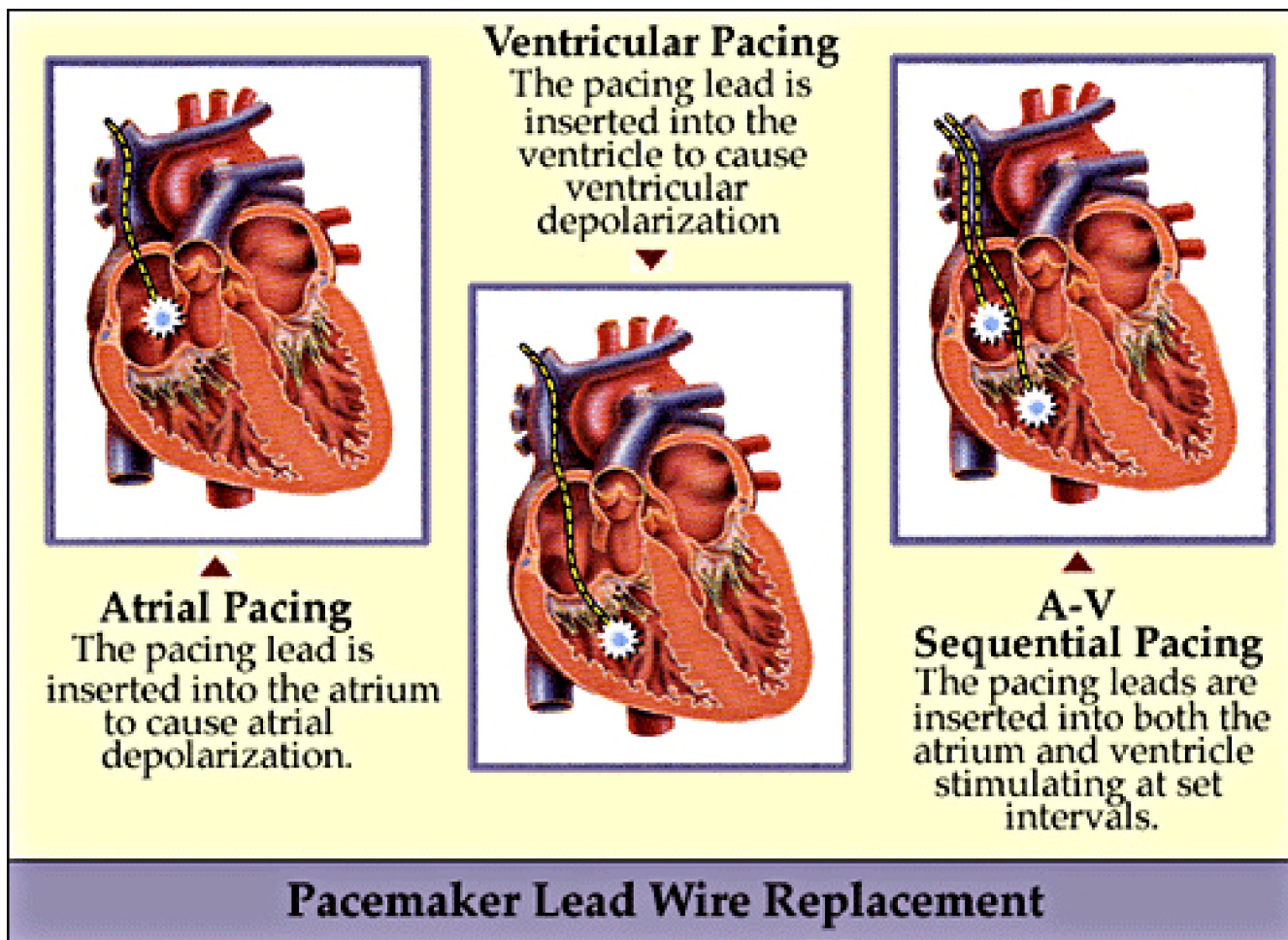


## Evolution of Acute MI

# Diagram: Stages of Acute Q-Wave MI-KH

*Frank G. Yanowitz, M.D.*





## Pacemaker Lead Wire Placement Diagram - Marquette-KH

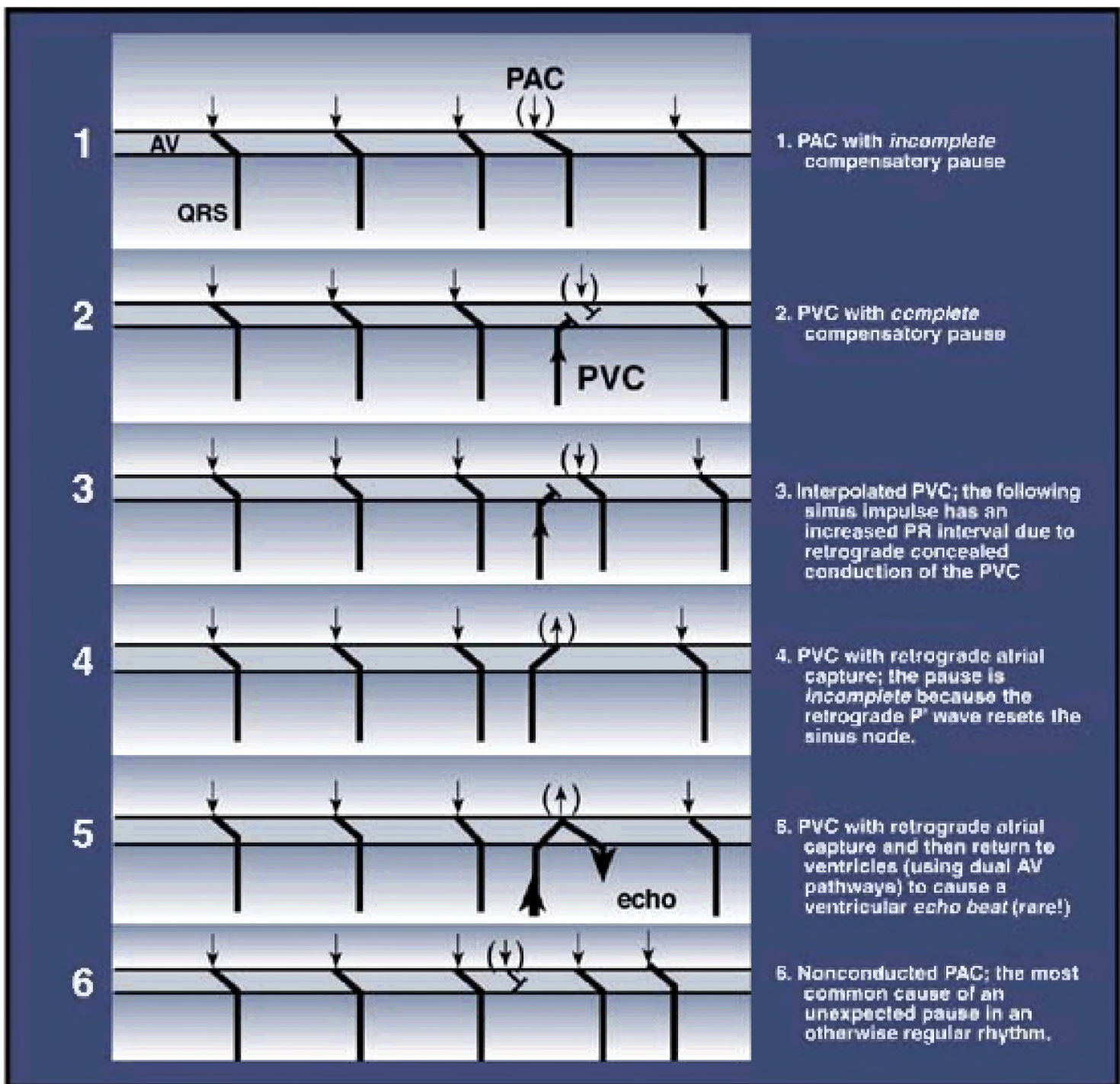
*Marquette Electronics Copyright 1996*

## **Dr. Alan Lindsay: A Teacher of Substance and Style**



## **Alan E. Lindsay, MD: A Teacher of Substance and Style**

*Frank Yanowitz Copyright 1996*

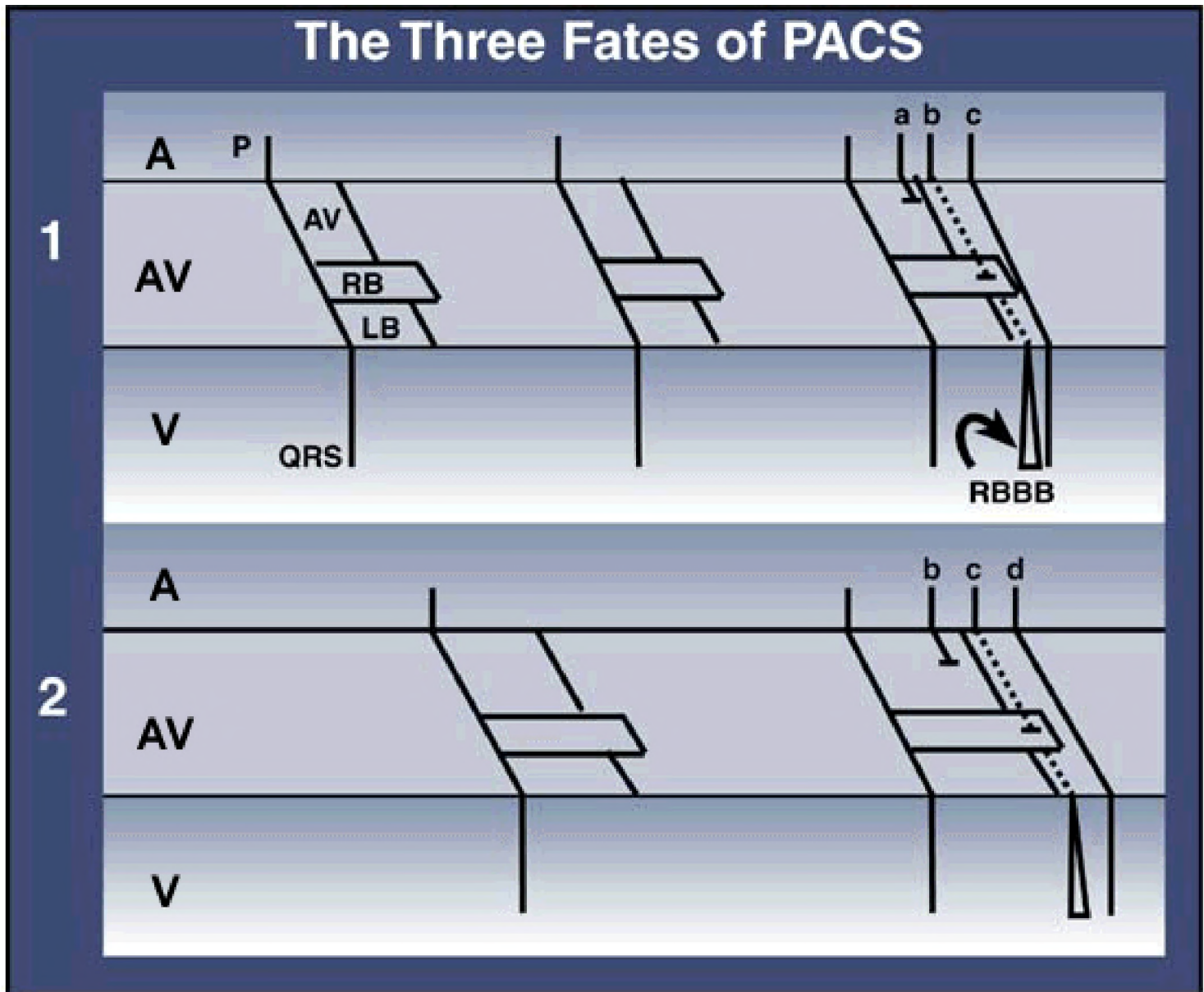


# All About Premature Beats-KH

Frank G. Yanowitz, M.D., copyright 1997

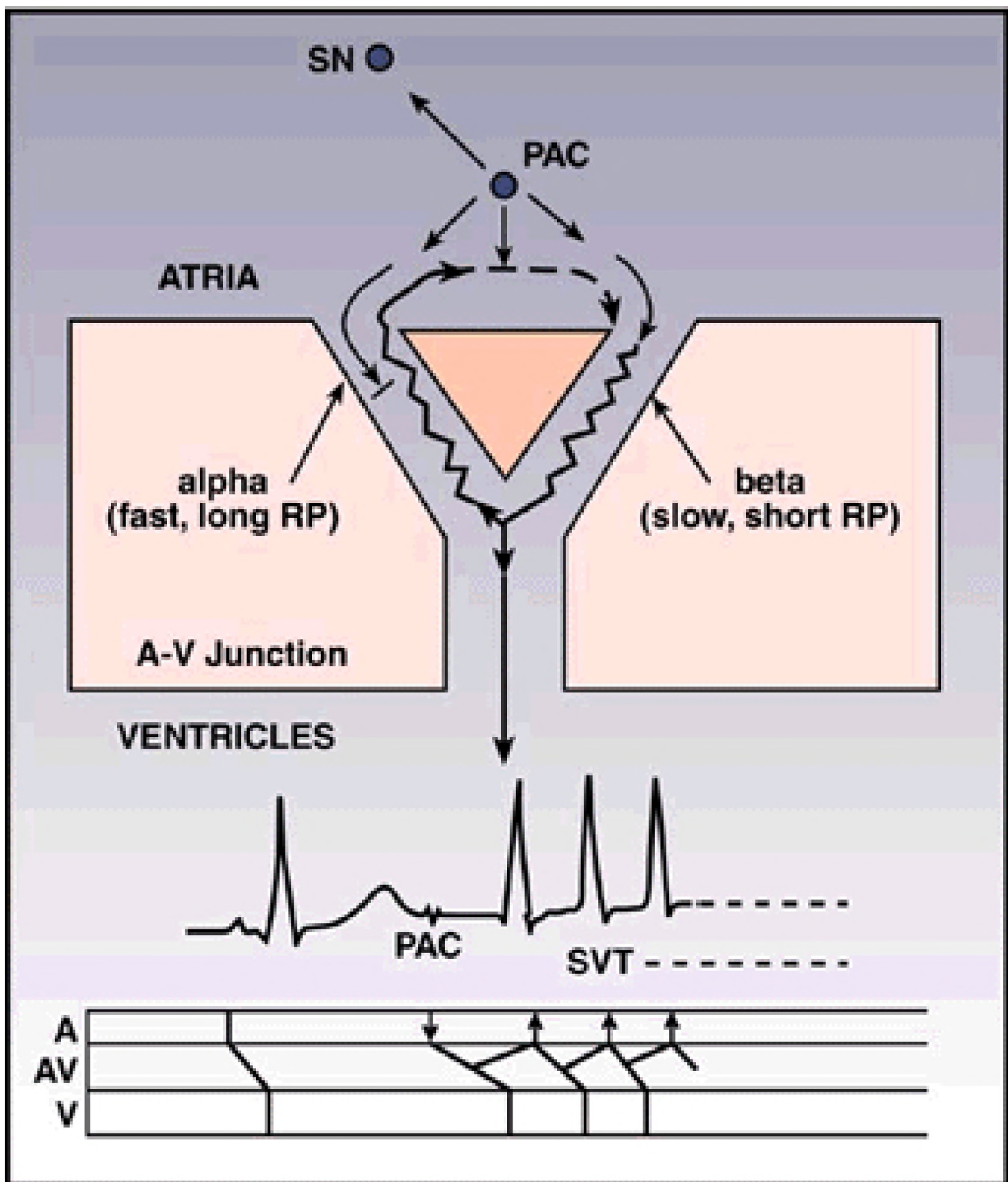


## The Three Fates of PACS



**The Three Fates Of PAC's: 1. Normal Conduction; 2. Aberrant Conduction; 3. Non-conduction-KH**

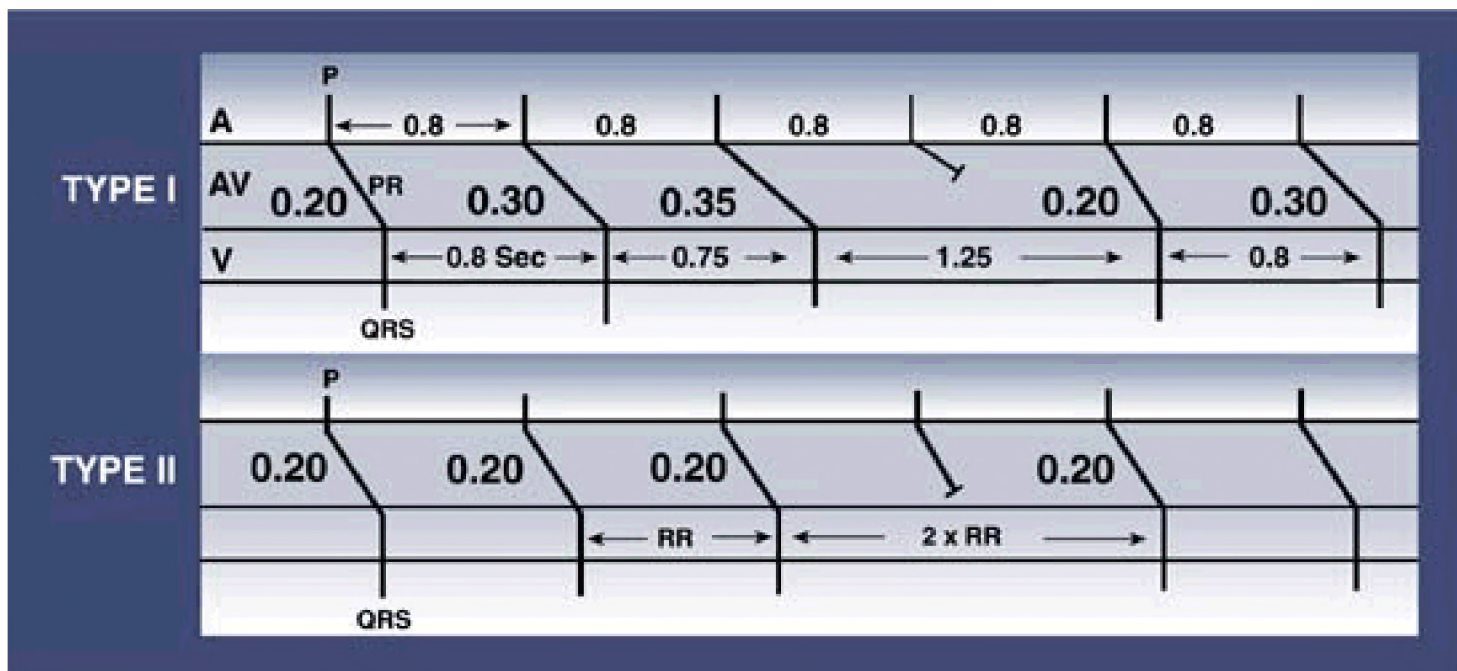
*Frank G. Yanowitz, M.D., copyright 1997*



**Diagram: AV Nodal Reentrant Tachycardia-KH**

*Frank G. Yanowitz, M.D., copyright 1997*

The AV node often has dual pathways; in this diagram the alpha pathway is fast, but has a long refractory period; the beta pathway is conducted more slowly, but recovers faster. In sinus rhythm the faster alpha pathway is used and accounts for the normal PR interval. When a PAC occurs, however, the impulse may find the alpha pathway refractory, but able to traverse the beta pathway. When the premature impulse reaches the intersection of the two pathways, alpha may be recovered and allow retrograde activation of the atria; this may enable a reentrant tachycardia to develop, as illustrated in the diagram.

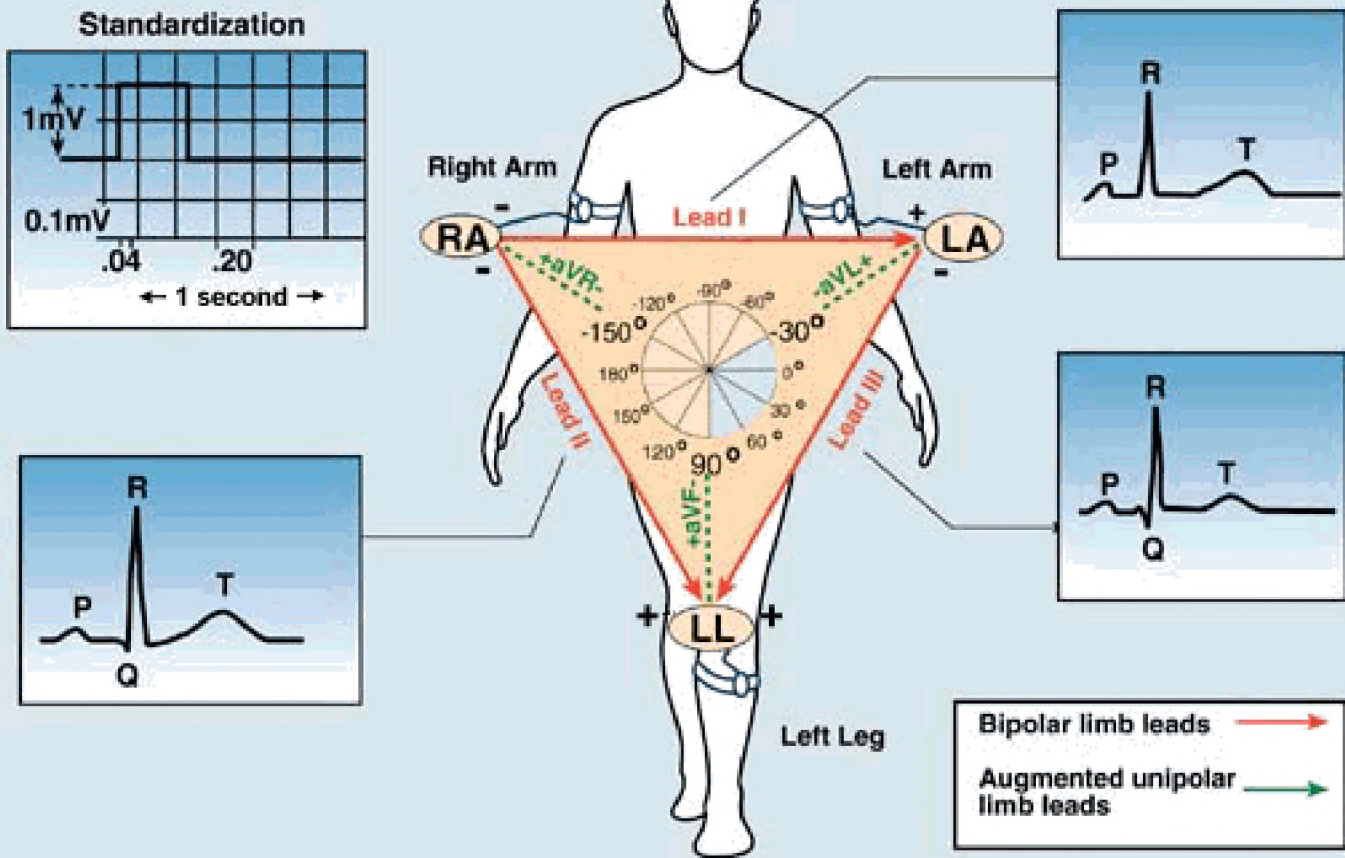


## Diagram: Type I vs. Type II 2nd Degree AV Block-KH

*Frank G. Yanowitz, M.D., copyright 1997*

In type I 2nd degree AV block the PR progressively lengthens until a nonconducted P wave occurs. The PR gets longer by smaller and smaller increments; this results in gradual shortening of the RR intervals. The RR interval of the pause is usually less than the two preceding RR intervals. The RR interval after the pause is longer than the RR interval just before the pause. In type II AV block, the PR is constant until the nonconducted P wave occurs. The RR interval of the pause is usually 2x the basic RR interval.

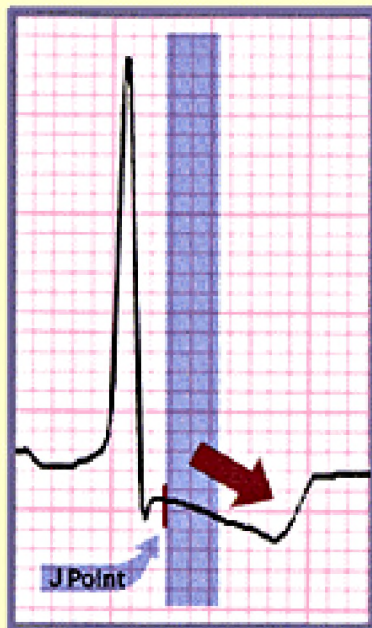
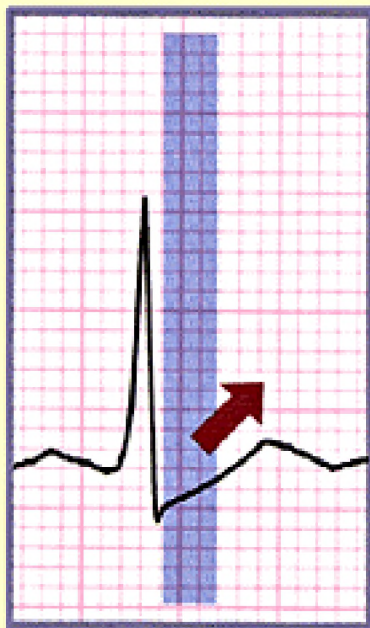
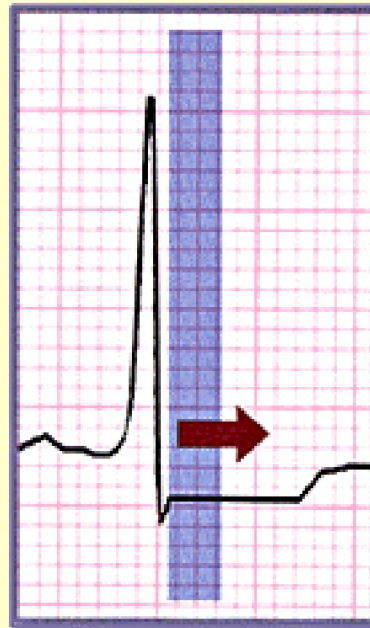
# The Standard Limb Leads



## Diagram: Frontal Plane Leads-KH

*Frank G. Yanowitz, M.D., copyright 1997*



**Downsloping ST****Upsloping ST****Horizontal ST**

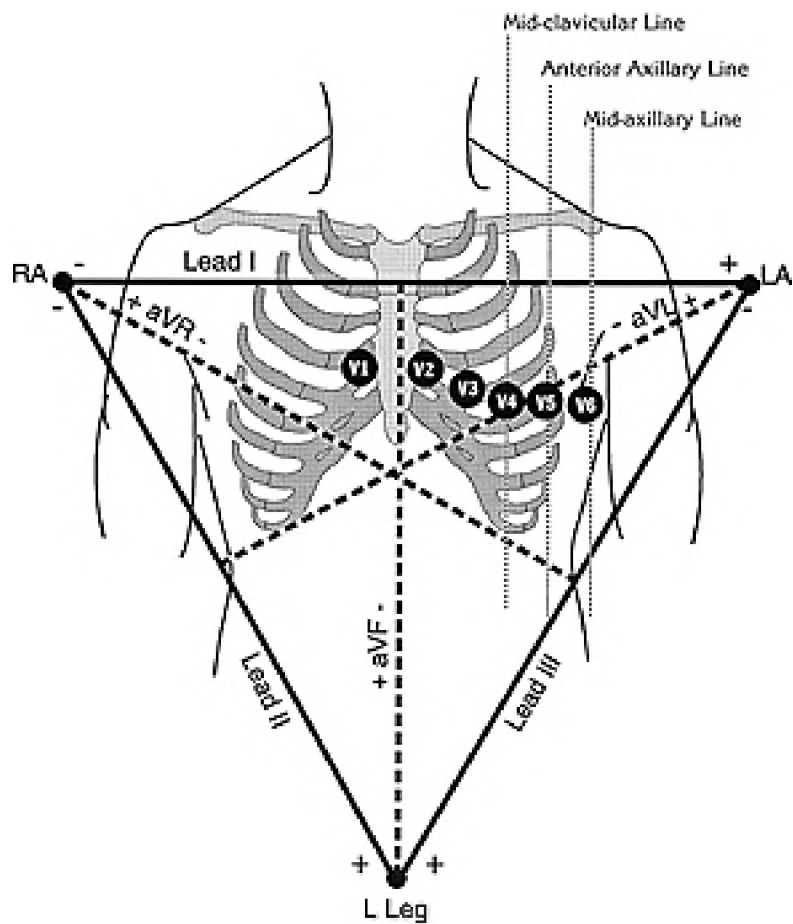
The J point occurs at the end of the QRS complex.  
The ST segment begins at the J point and extends to a user defined interval

## ST Segment Depression

# ST Segment Diagram - Marquette-KH

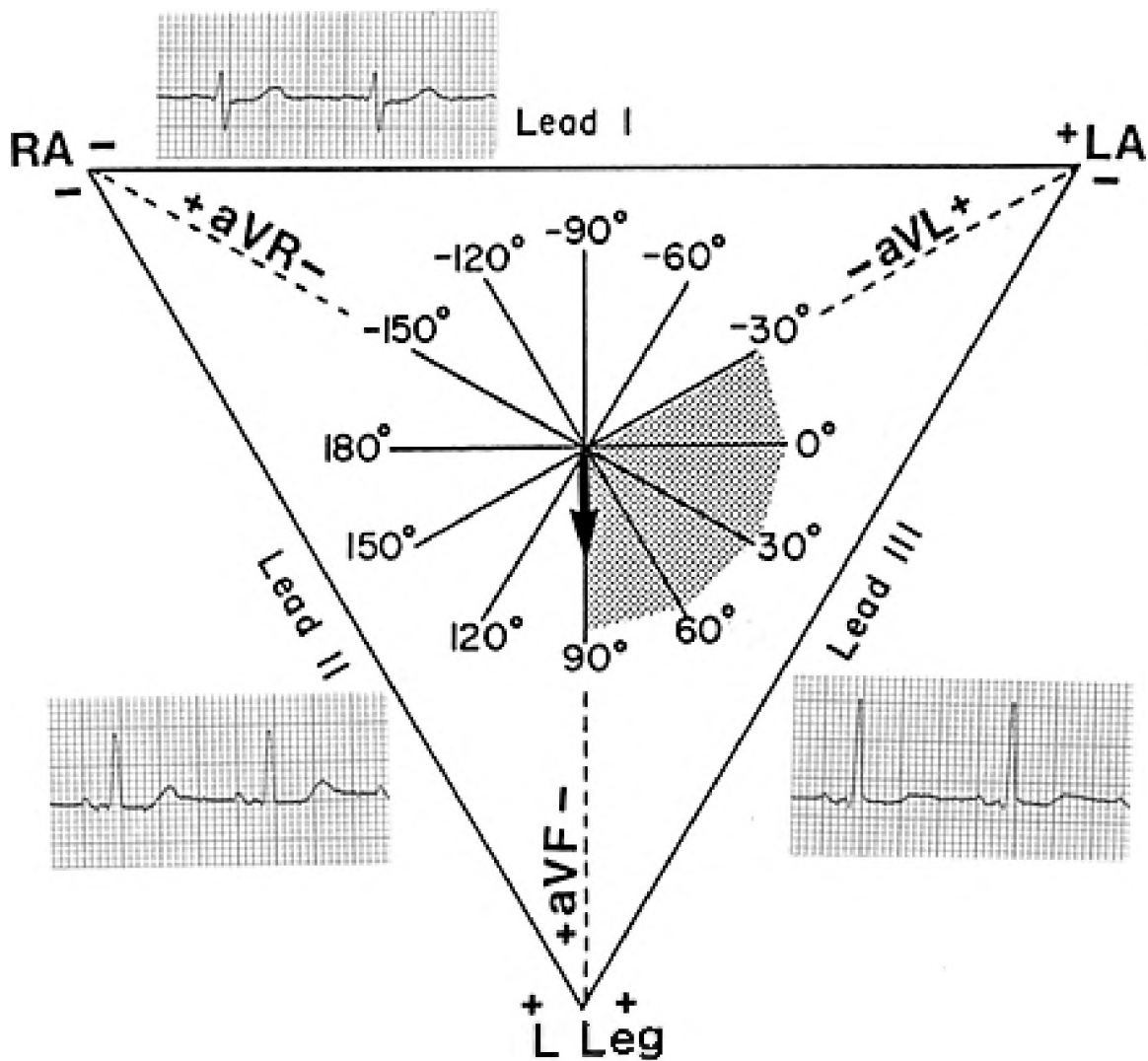
*Marquette Electronics Copyright 1996*





# Frontal and Horizontal Plane Lead Diagram-KH

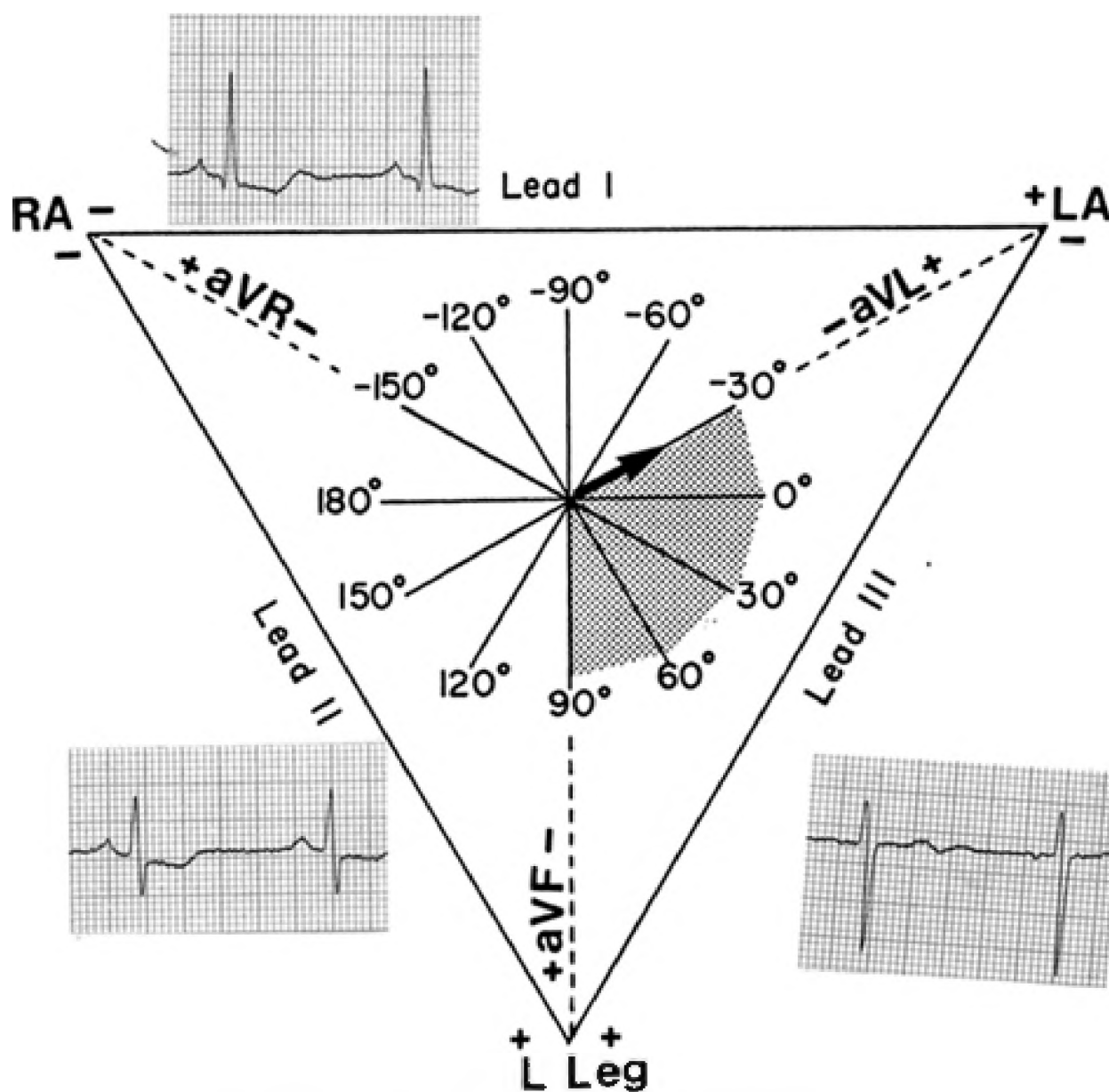
*Frank G. Yanowitz, M.D.*



**QRS Axis = +90 degrees-KH**

*Frank Yanowitz Copyright 1996*

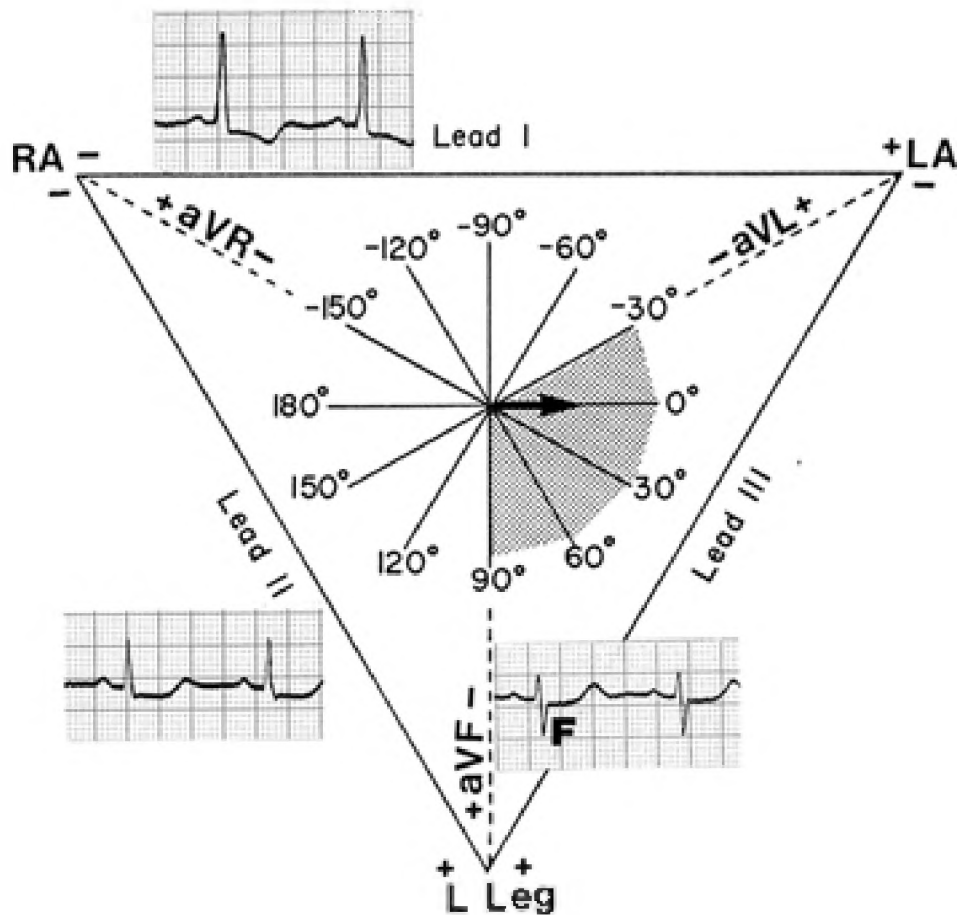
Lead I is isoelectric; II and III are positive; the axis is +90 degrees.



**QRS Axis = -30 degrees-KH**

*Frank Yanowitz Copyright 1996*

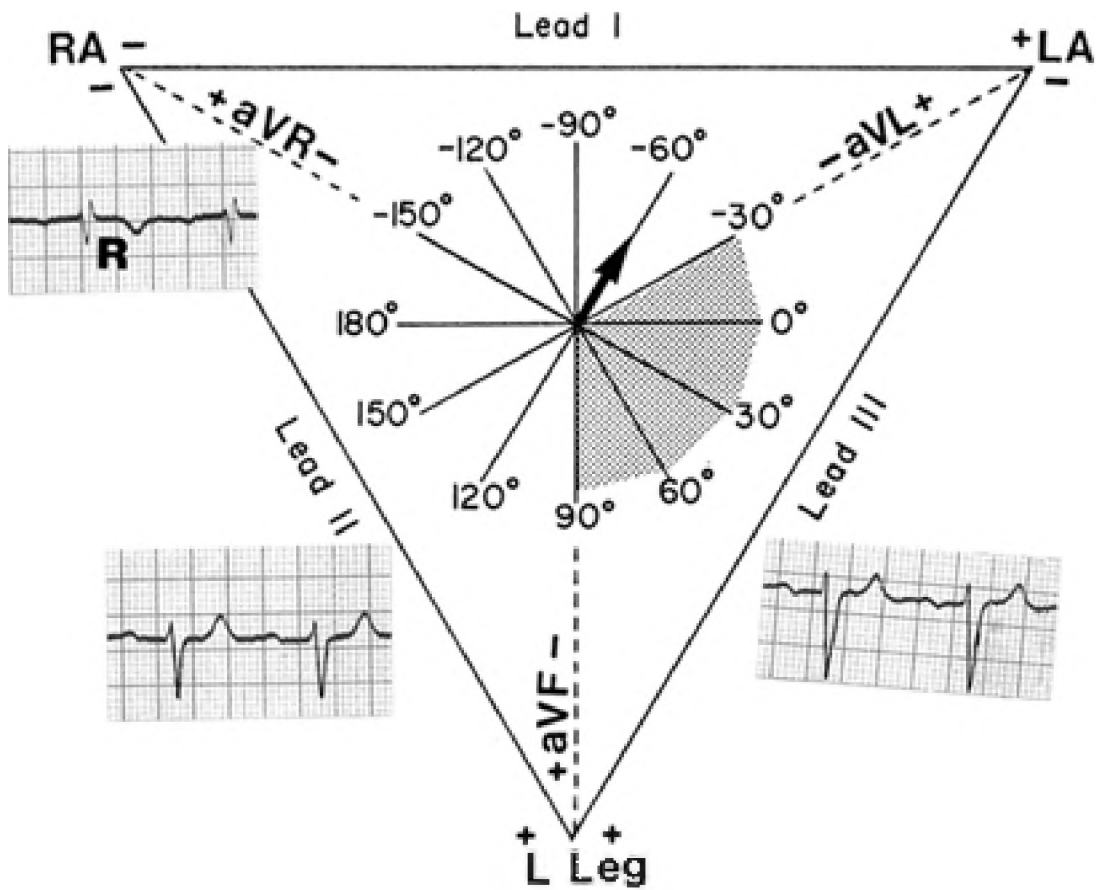
Lead II is isoelectric; I is positive; III is negative. The axis is -30 degrees.



## QRS Axis = 0 degrees-KH

*Frank Yanowitz Copyright 1996*

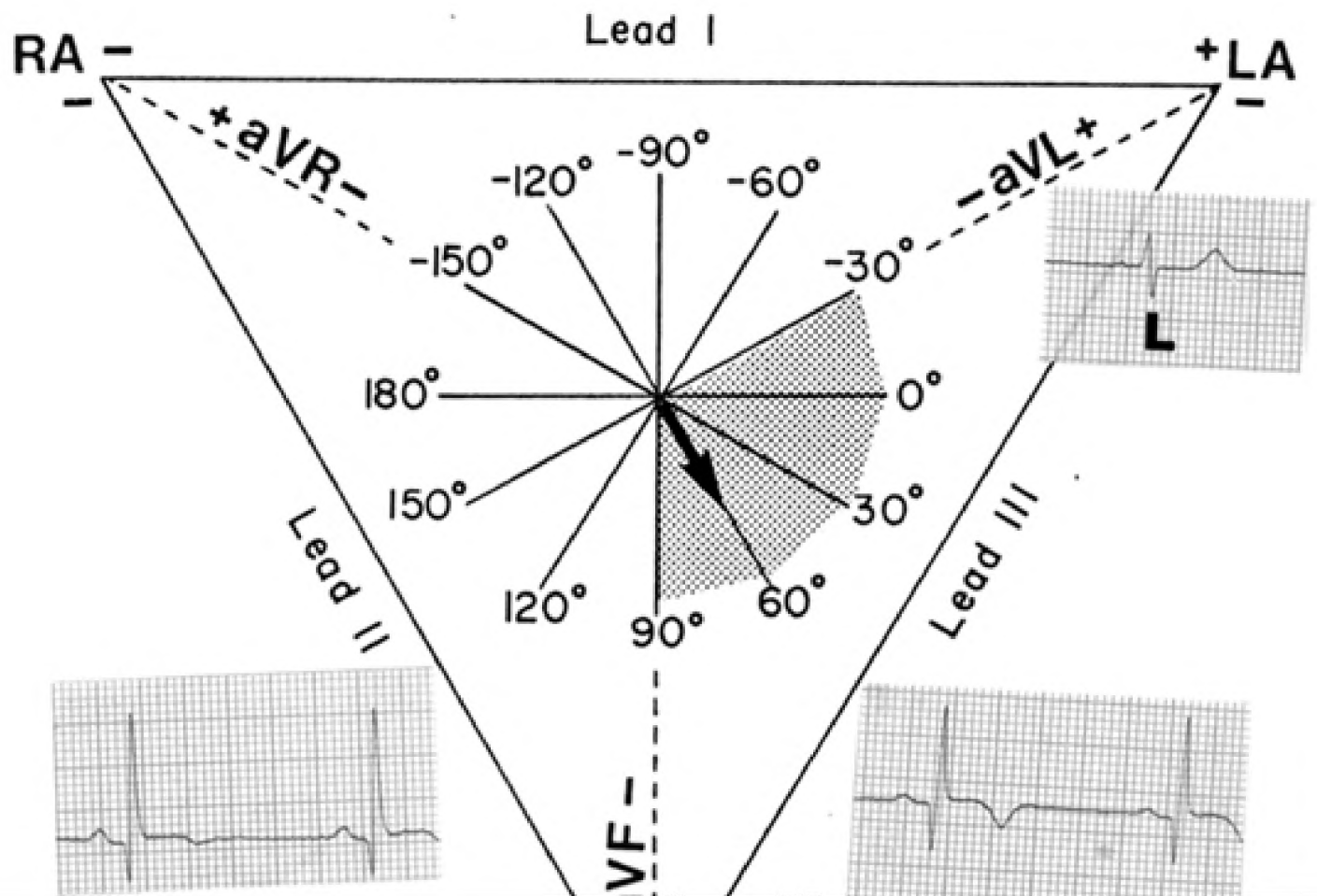
Lead aVF is isoelectric; lead I is positive; therefore, the QRS axis is 0 degrees.



## Left Axis Deviation: QRS Axis = -60 degrees-KH

*Frank Yanowitz Copyright 1996*

Lead aVR is isoelectric; leads II and III are mostly negative. The QRS axis, therefore, is -60 degrees.

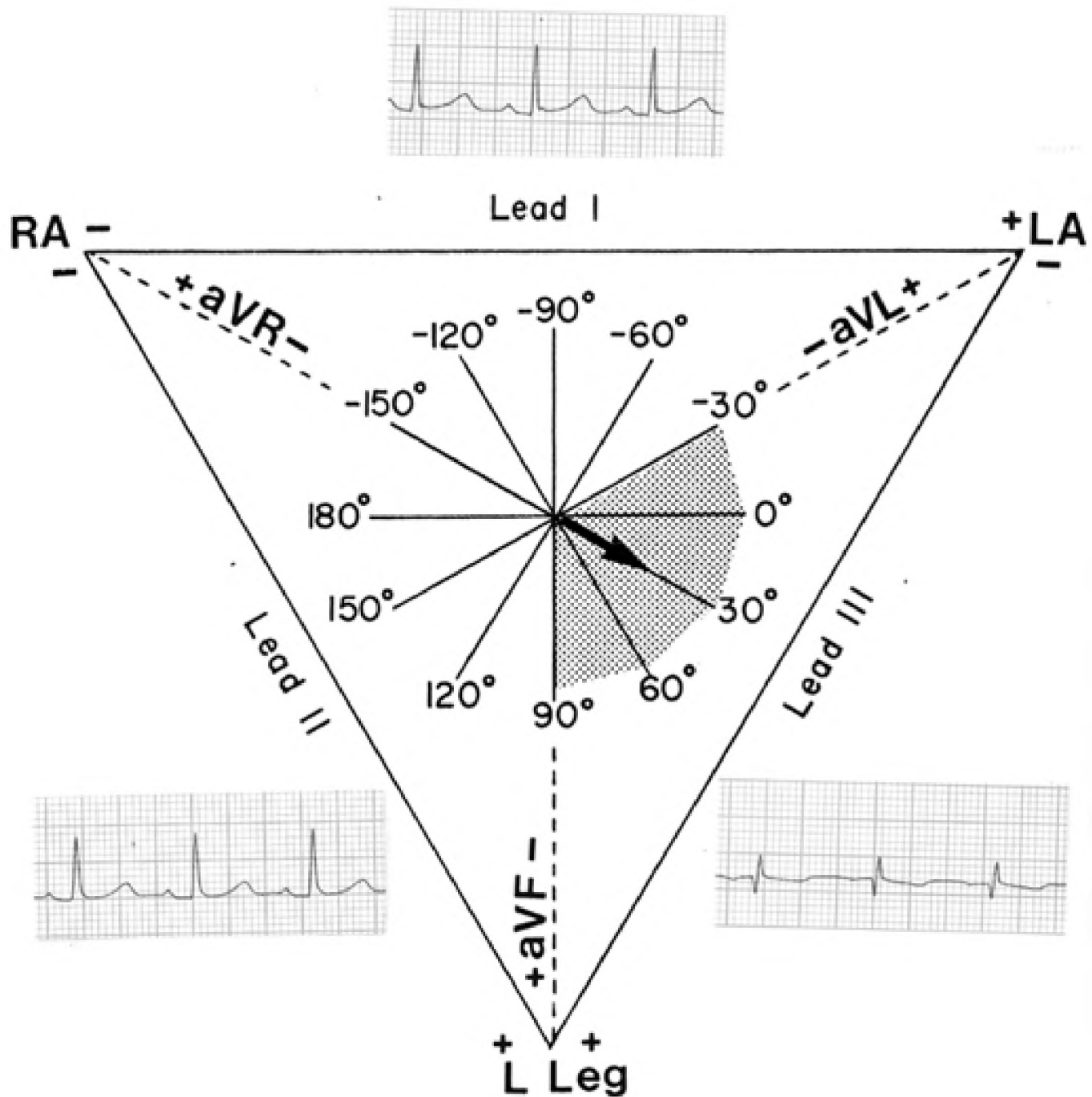


**QRS Axis = +60 degrees-KH**

*Frank Yanowitz Copyright 1996*

Lead aVL is isoelectric; leads II and III are mostly positive. The QRS axis, therefore, is +60 degrees.



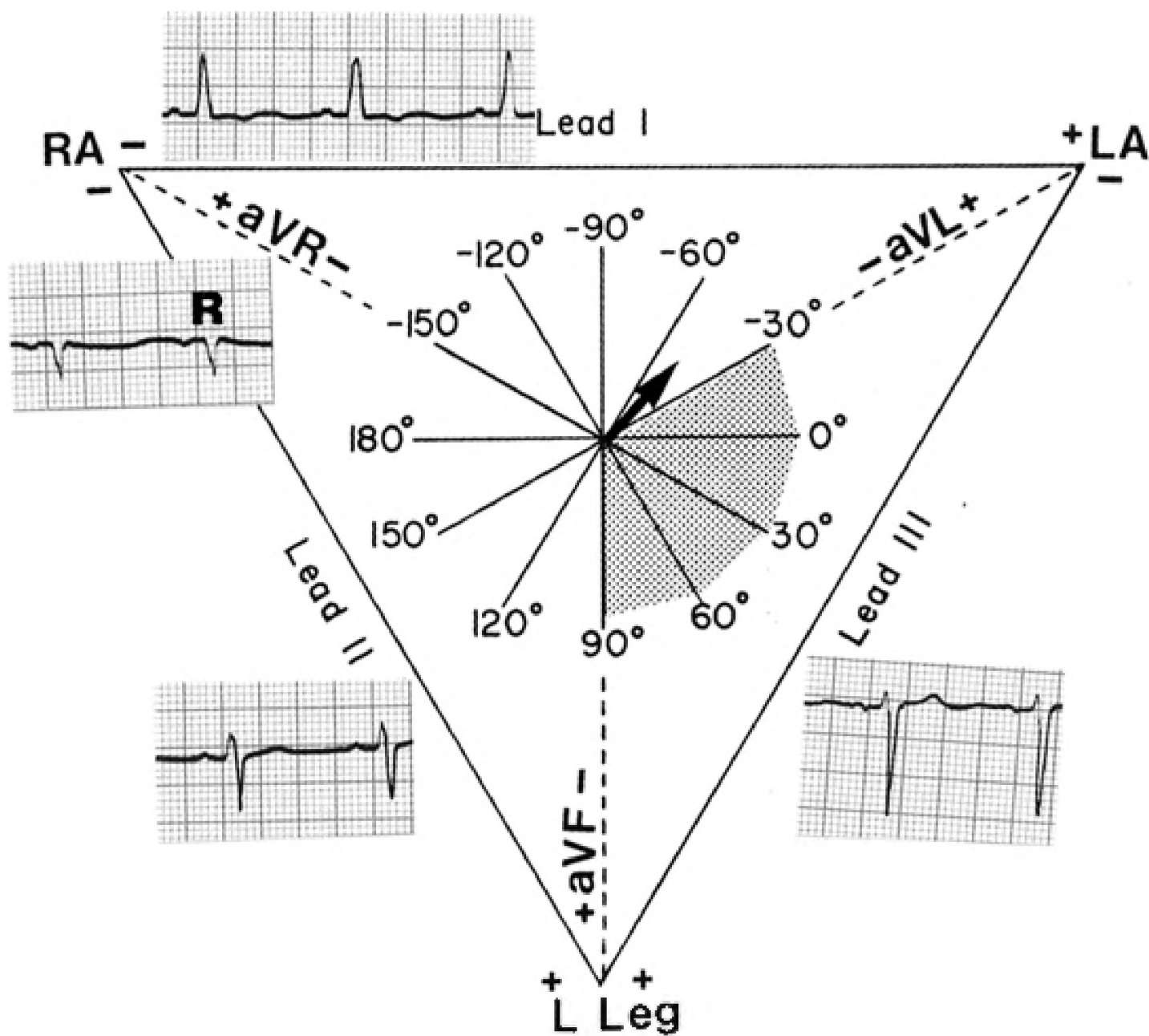


**QRS Axis = +30 degrees-KH**

*Frank Yanowitz Copyright 1996*

Lead III is isoelectric; leads I and II are positive. The QRS axis, therefore, is +30 degrees.

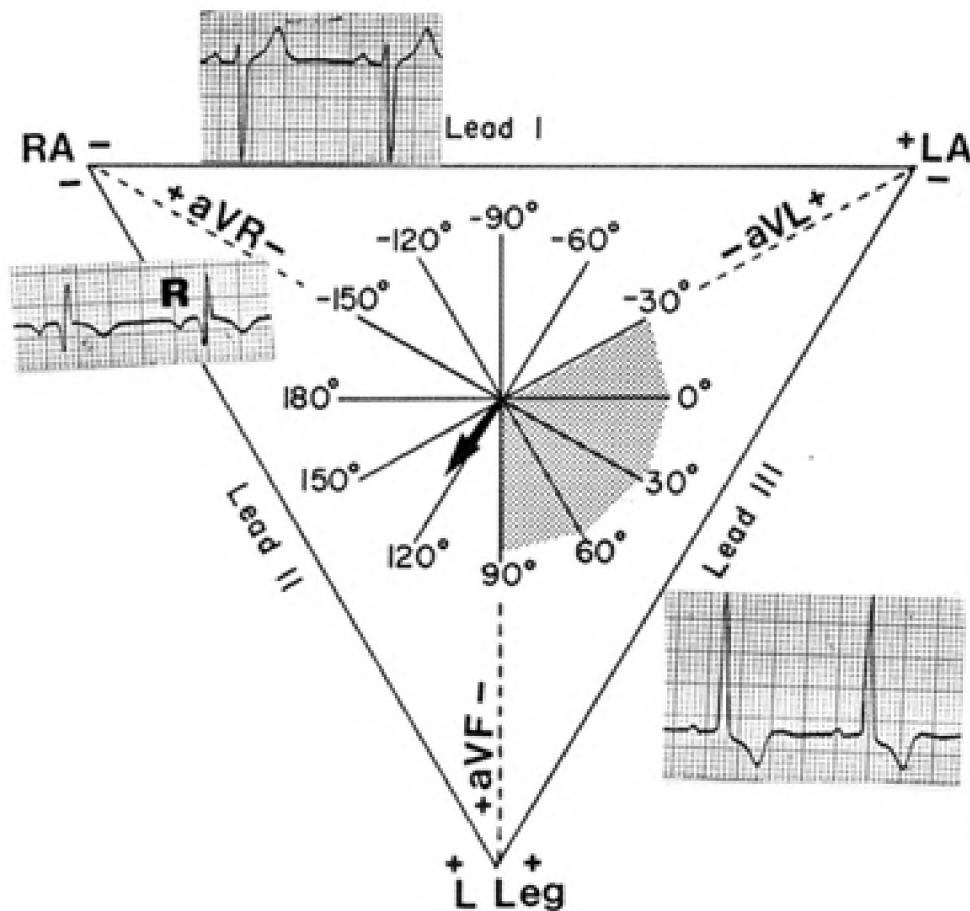




## Left Axis Deviation: QRS Axis = -45 degrees-KH

*Frank Yanowitz Copyright 1996*

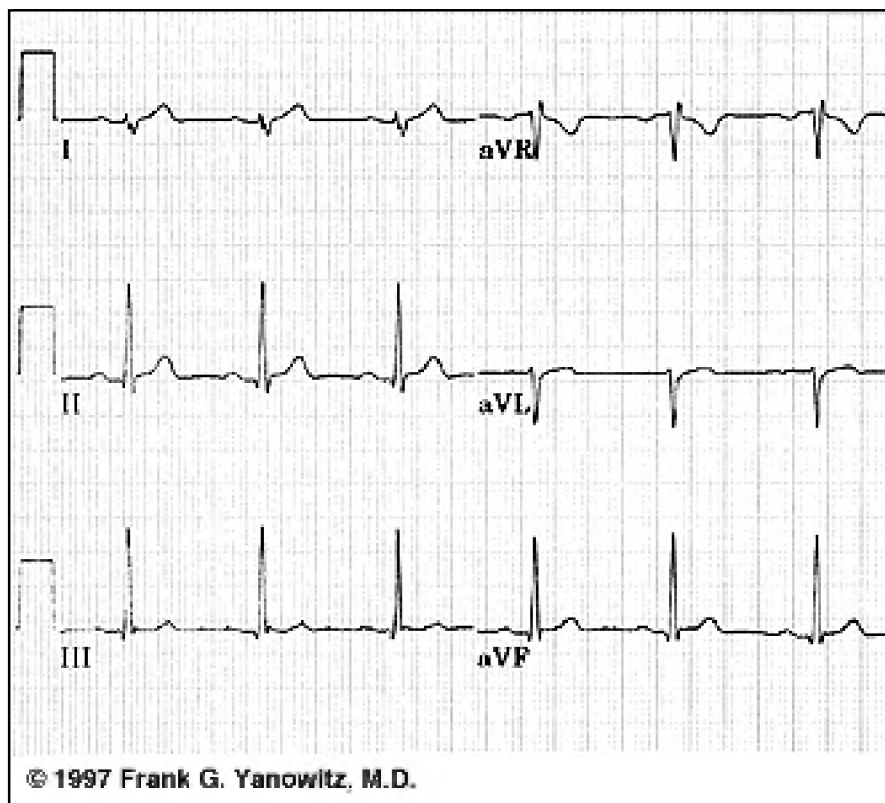
There is no isoelectric, but leads aVR and II are the closest to being isoelectric, placing the axis between -30 and -60 degrees. The axis, therefore, is about -45 degrees.



## Right Axis Deviation: QRS Axis = +130 degrees-KH

*Frank Yanowitz Copyright 1996*

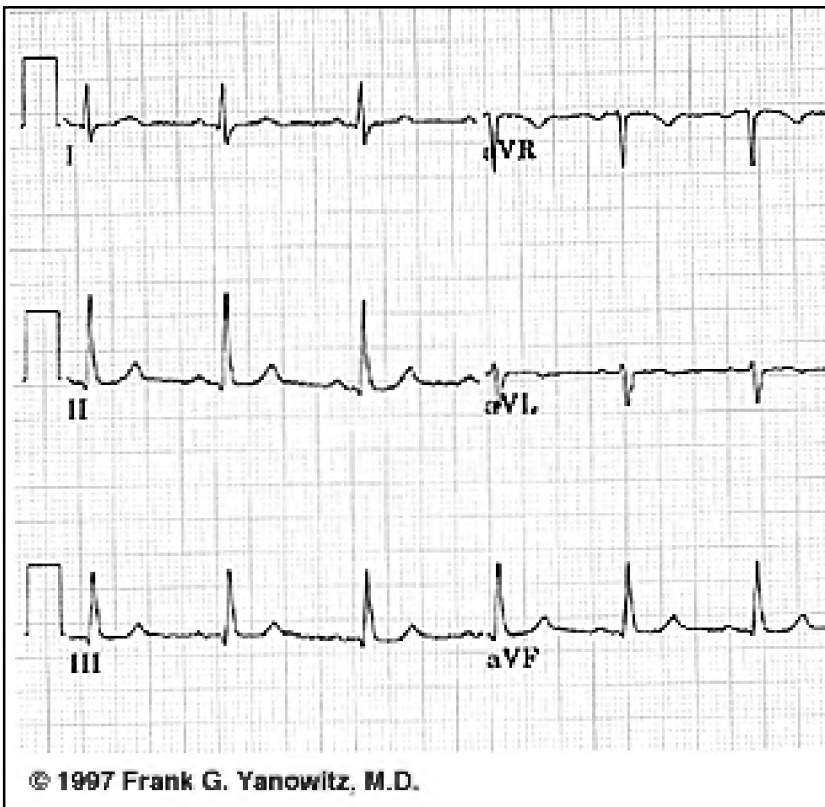
Lead aVR is almost isoelectric; lead I is mostly negative, and lead III is very positive. The QRS axis, therefore, is +130 degrees. Note that the slightly more positive AVR moves the axis slightly beyond +120 degrees; i.e., closer to the + pole of the aVR lead.



## Frontal Plane QRS Axis = +90 degrees-KH

*Frank G. Yanowitz, M.D.*

1) Lead I is isoelectric; 2) perpendiculars to lead I are +90 and -90 degrees; 3) leads II, III, aVF are positive; 4) therefore, the axis must be +90 degrees.

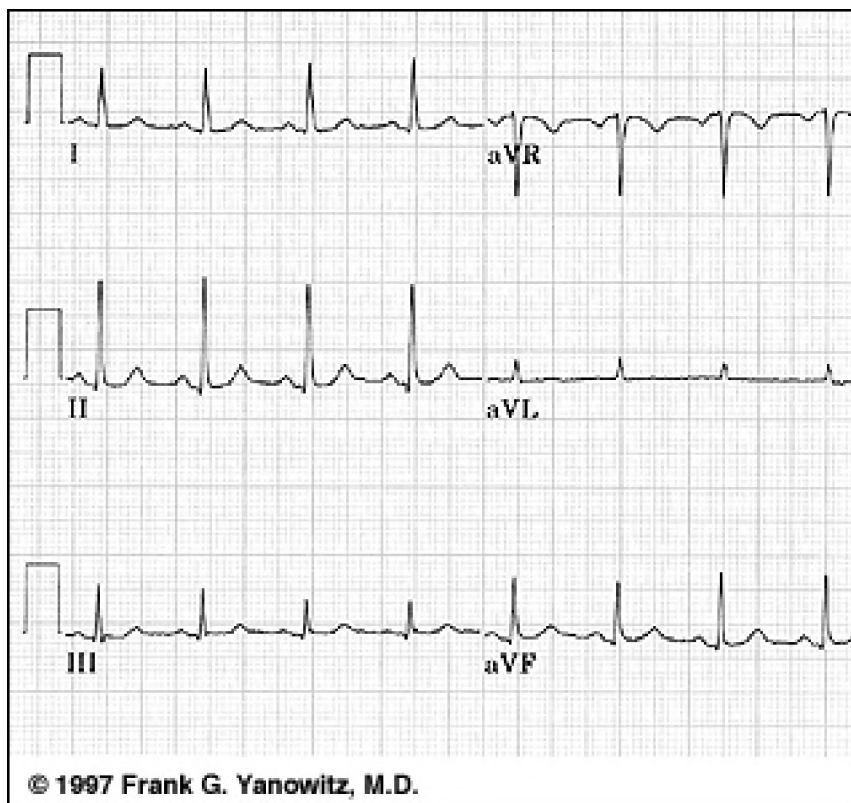


## Frontal Plane QRS Axis = +75 degrees-KH

*Frank G. Yanowitz, M.D.*

Since there is no isoelectric lead in this ECG, the two closest leads are I and aVL. If I were isoelectric, the axis would be +90 degrees; if aVL were isoelectric, the axis would be +60 degrees. A nice compromise is +75 degrees. (The two closest leads are always 30 degrees apart.)

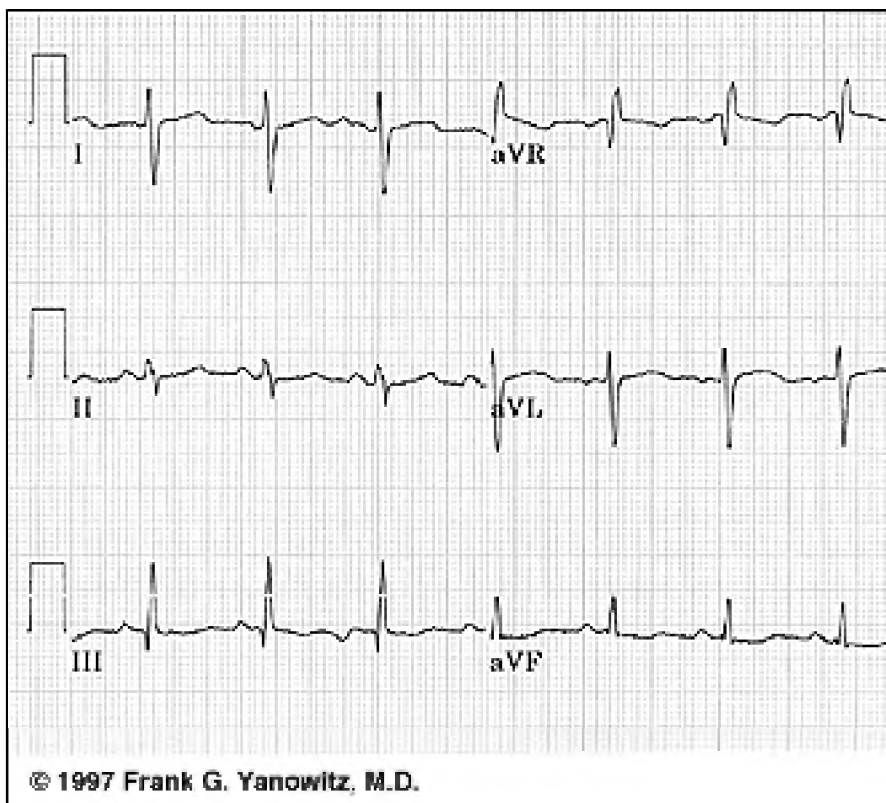




## Frontal Plane QRS Axis = +50 degrees-KH

*Frank G. Yanowitz, M.D.*

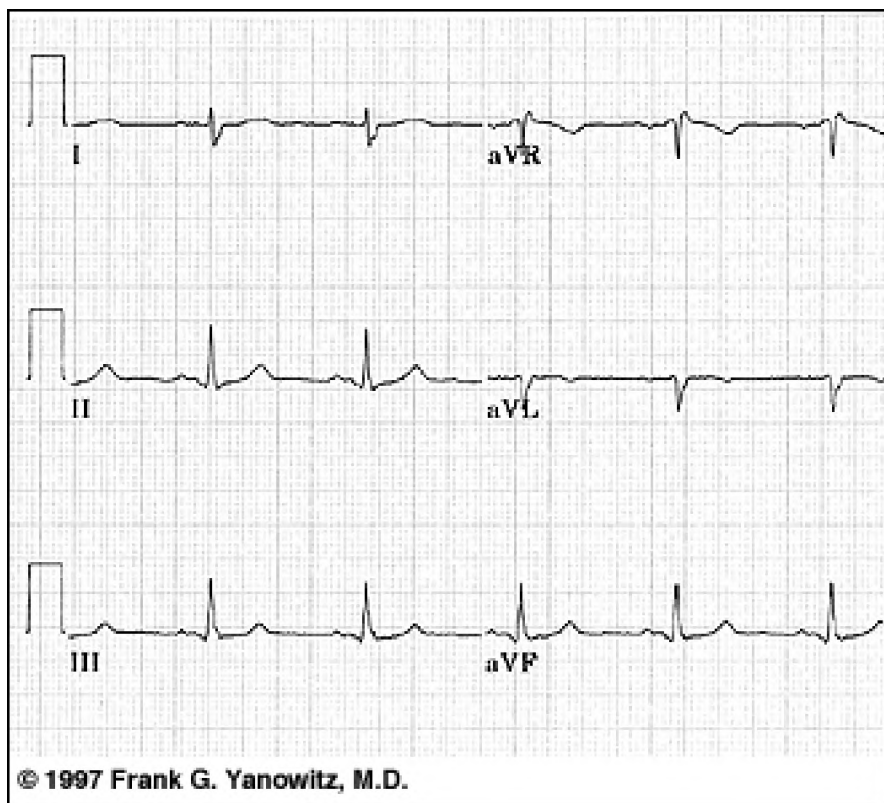
1) lead aVL is the smallest QRS and closest to being the isoelectric lead; 2) perpendiculars to aVL are +60 and -120 degrees; 3) lead I is positive; 4) therefore, the axis is closest to being +60 degrees. Because aVL is actually slightly positive, the axis is only about +50 degrees (i.e., slightly to the left of +60).



## Frontal Plane QRS Axis = +150 degrees (RAD)-KH

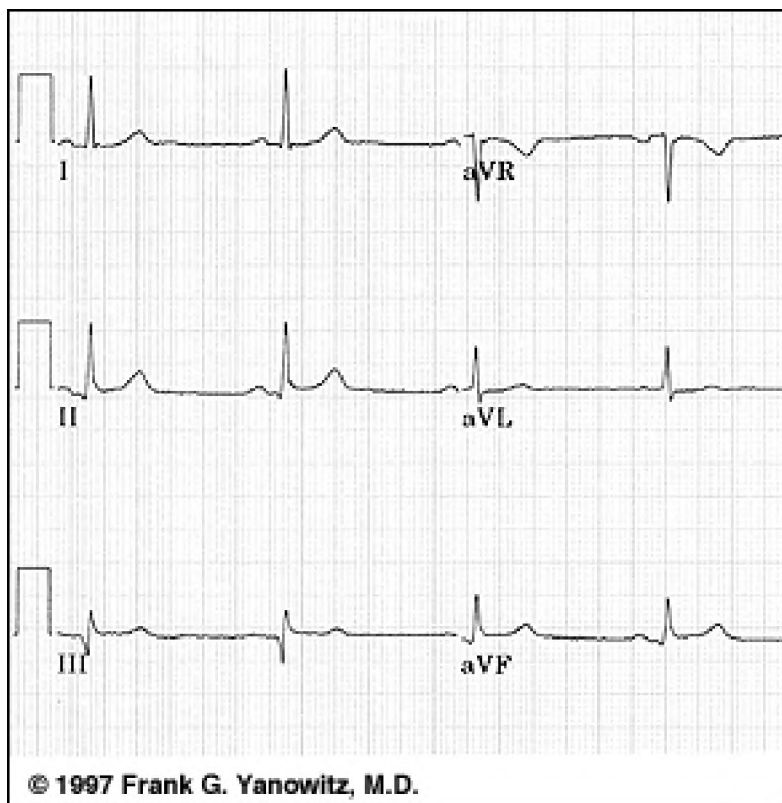
*Frank G. Yanowitz, M.D.*

This is an unusual right axis deviation (RAD). Lead I is negative, which usually means RAD. Lead II is the isoelectric lead, which almost always means -30 degrees; but in this example the axis is 180 degrees away from -30, or +150 degrees.



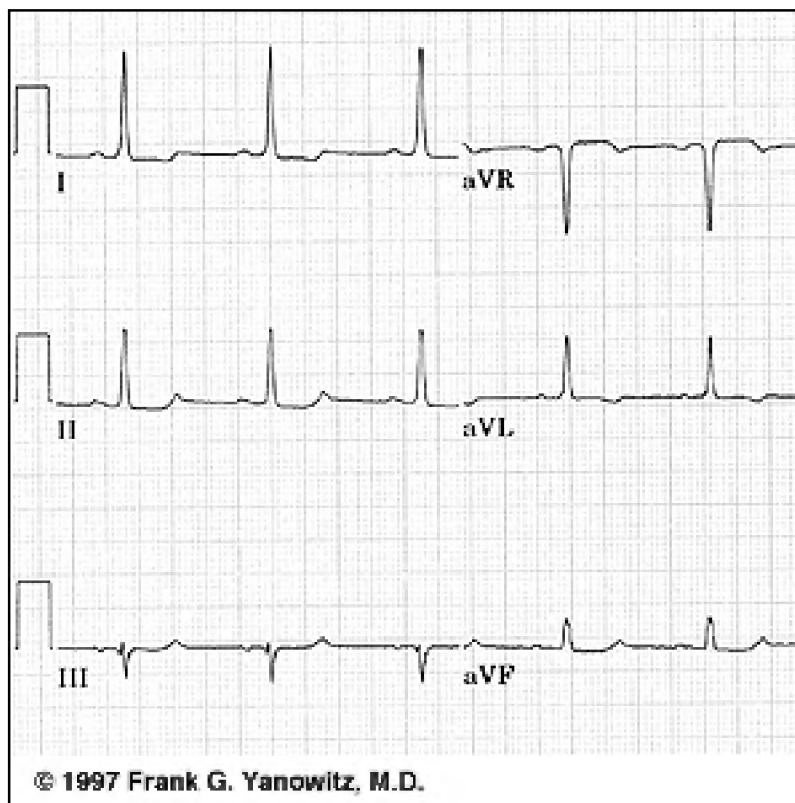
**Frontal Plane QRS Axis = 90 degrees-KH**

*Frank G. Yanowitz, M.D.*



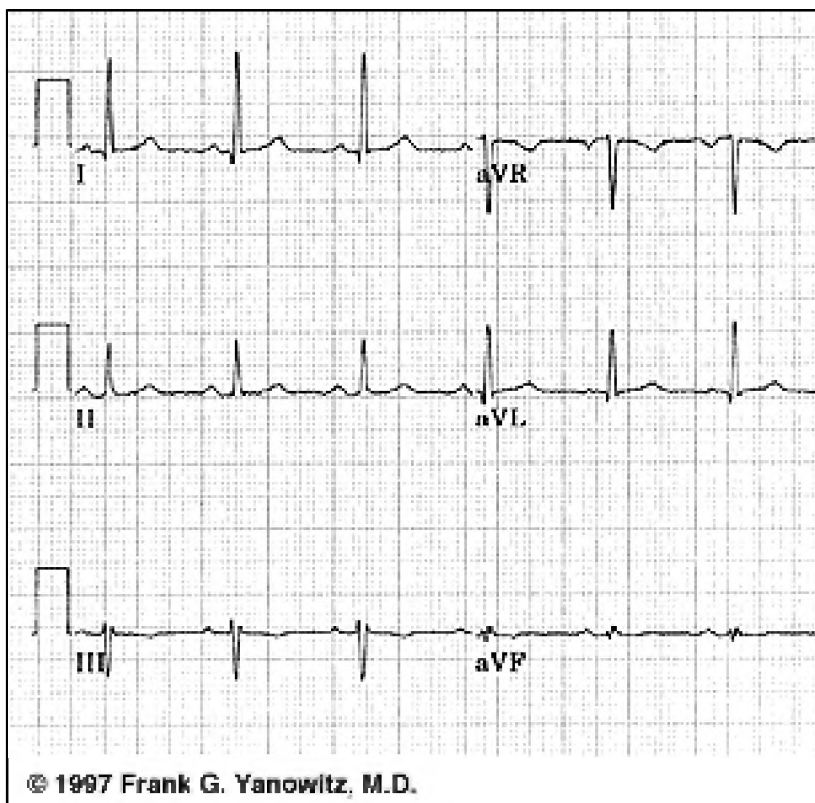
**Frontal Plane QRS Axis = +30 degrees-KH**

*Frank G. Yanowitz, M.D.*



**Frontal Plane QRS Axis = +15 degrees-KH**

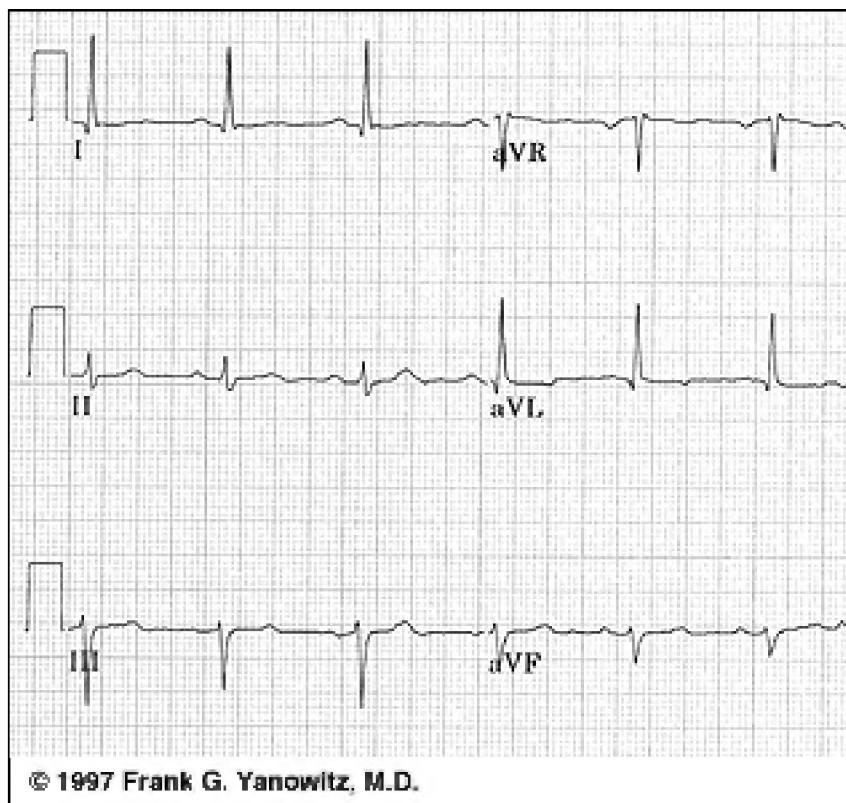
*Frank G. Yanowitz, M.D.*



**Frontal Plane QRS Axis = 0 degrees-KH**

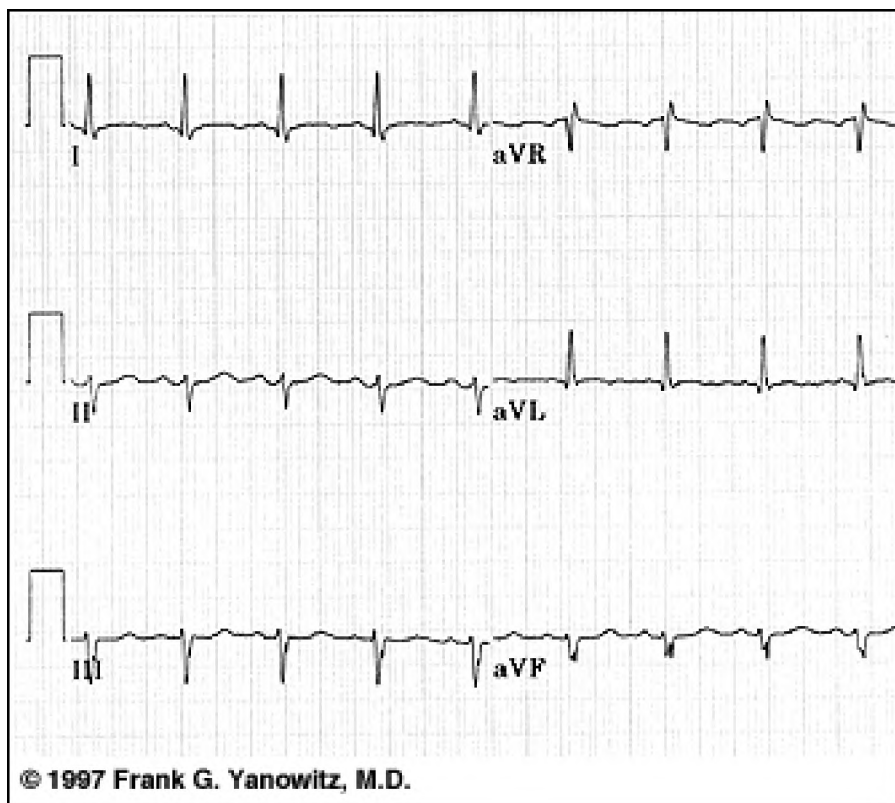
*Frank G. Yanowitz, M.D.*





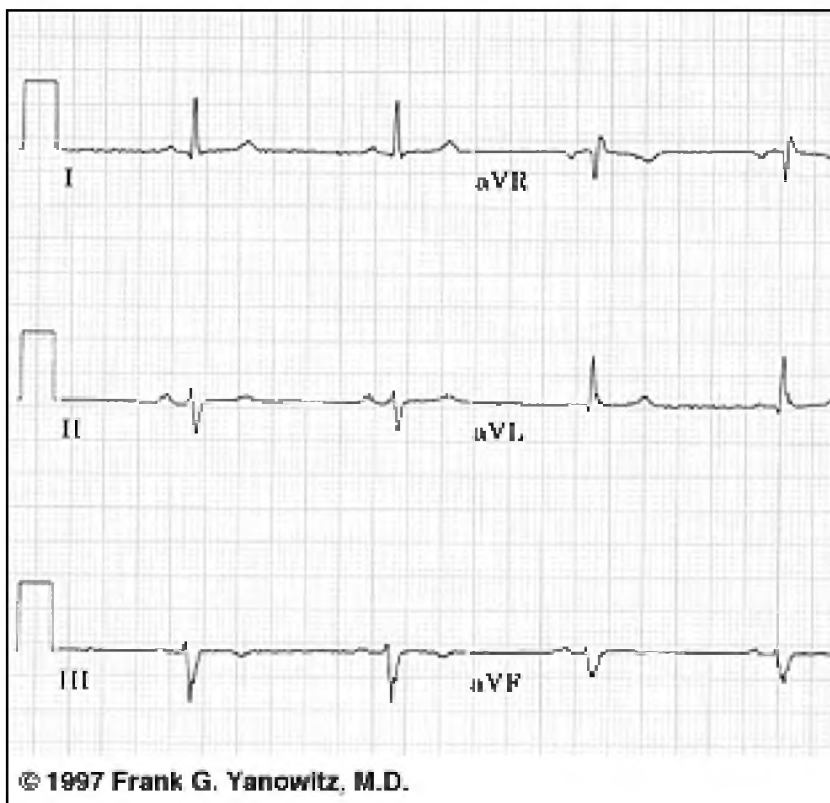
**Frontal Plane QRS Axis = -15 degrees-KH**

*Frank G. Yanowitz, M.D.*



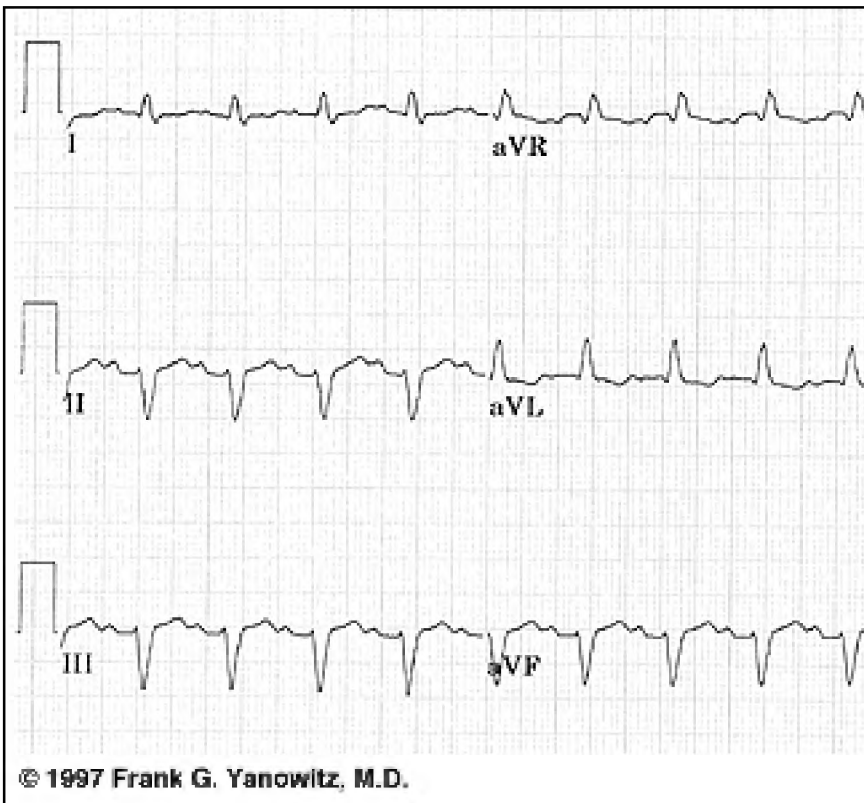
**Frontal Plane QRS Axis = -45 degrees-KH**

*Frank G. Yanowitz, M.D.*



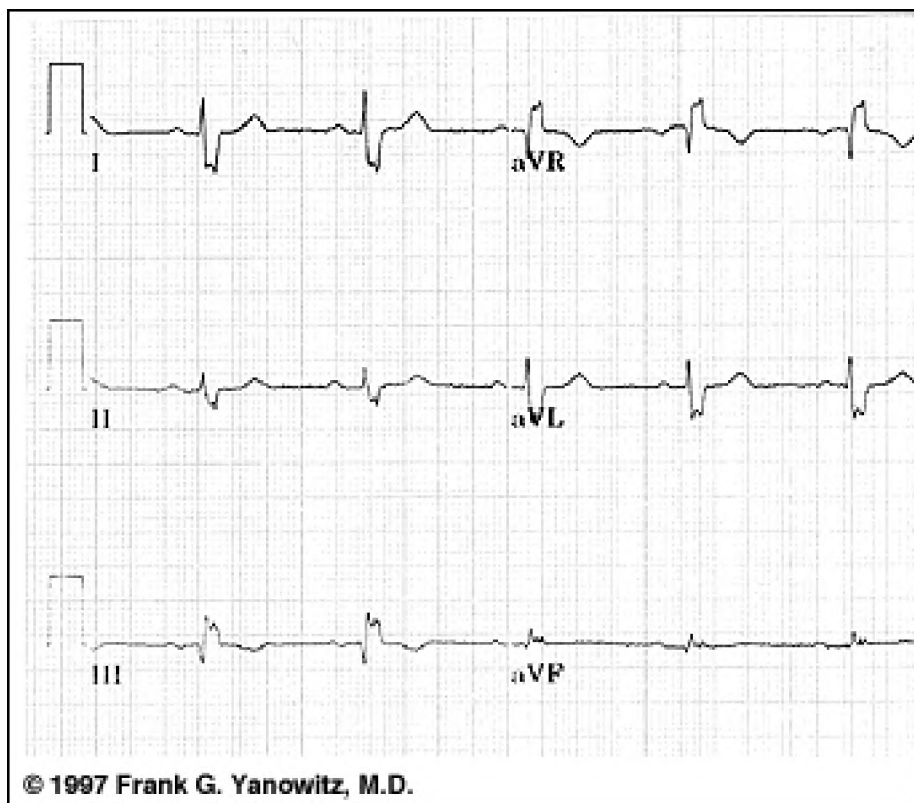
**Frontal Plane QRS Axis = -45 degrees-KH**

*Frank G. Yanowitz, M.D.*



**Frontal Plane QRS Axis = -75 degrees-KH**

*Frank G. Yanowitz, M.D.*



## Indeterminate Frontal Plane QRS Axis-KH

*Frank G. Yanowitz, M.D.*

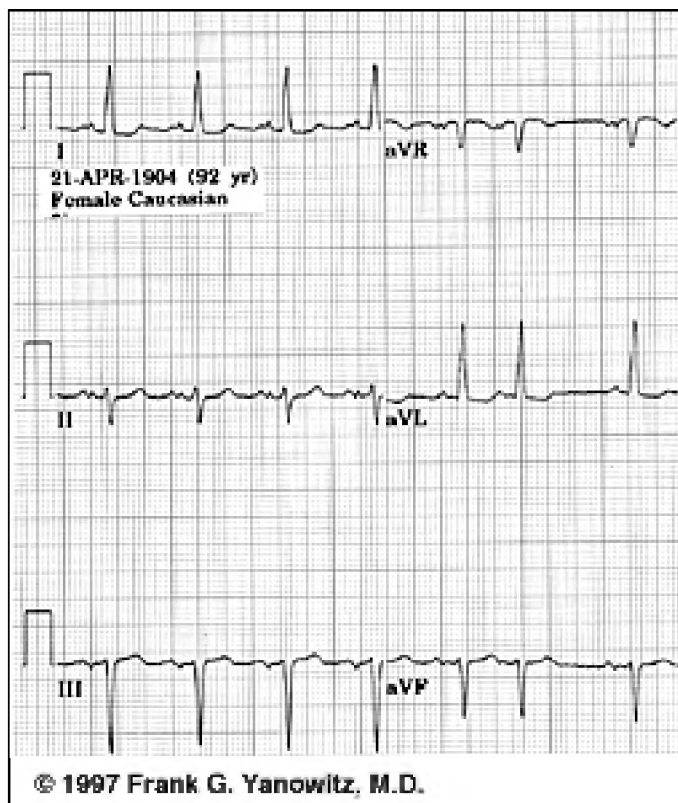


## Right Axis Deviation

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The isoelectric lead is approximately aVR; Because Lead I is more negative than positive, the axis is approximately +120 degrees

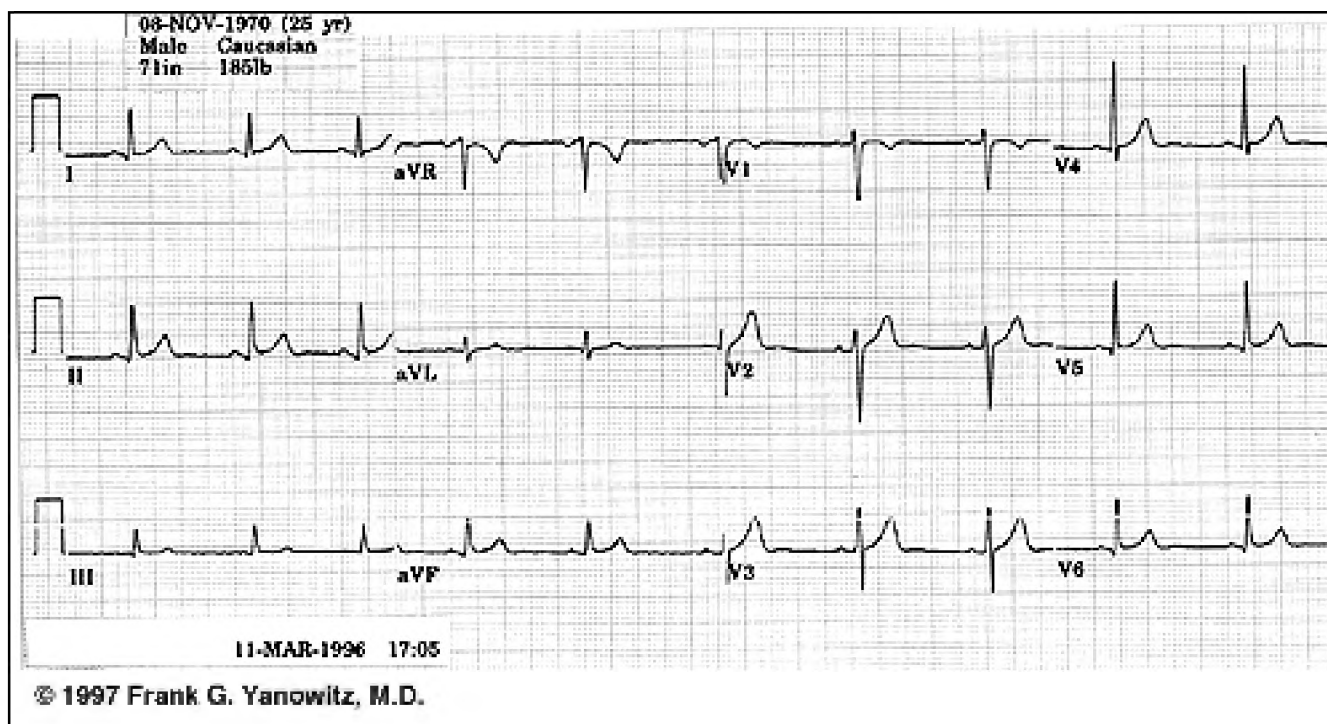




## Left Axis Deviation

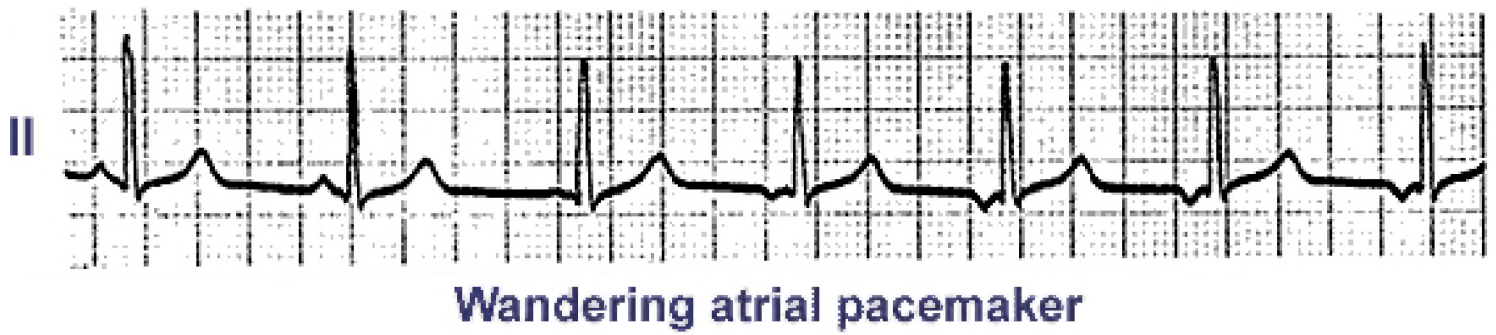
*Frank G. Yanowitz, M.D. Copyright 1998*

Lead II is more negative than positive, making the QRS axis more negative than -30 degrees. Because aVR is still negative, however, the axis is about -40 degrees. A PAC is also present.



## Normal ECG-KH

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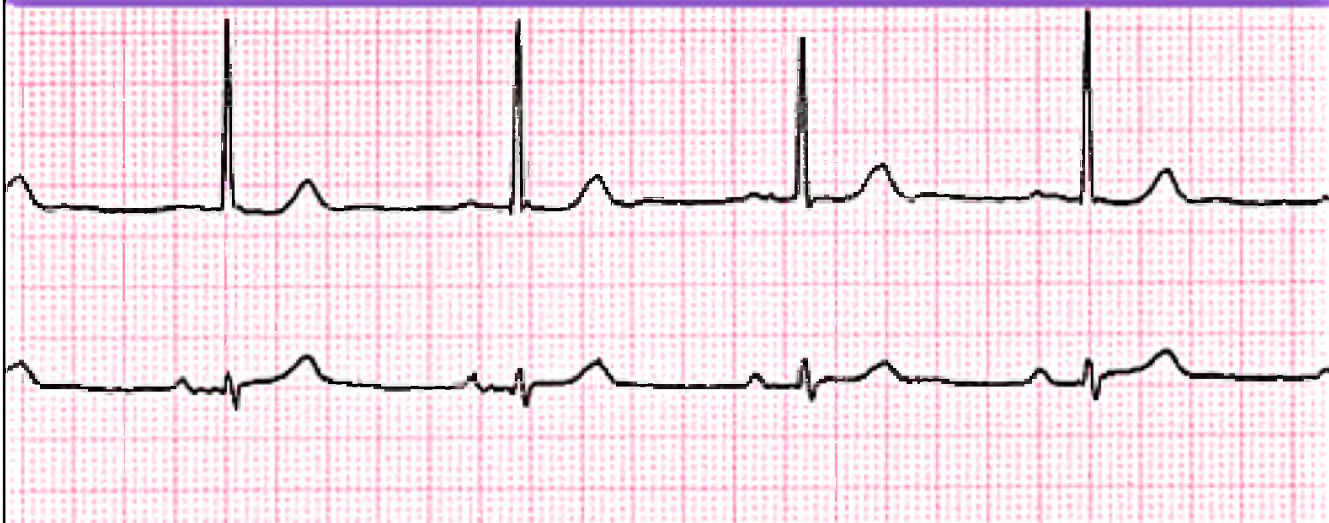


## Wandering Atrial Pacemaker-KH

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Wandering atrial pacemaker is a benign rhythm change where the pacemaker site shifts from the sinus node into the atrial tissues. P-wave morphology varies with the pacemaker site.

## Sinus Bradycardia

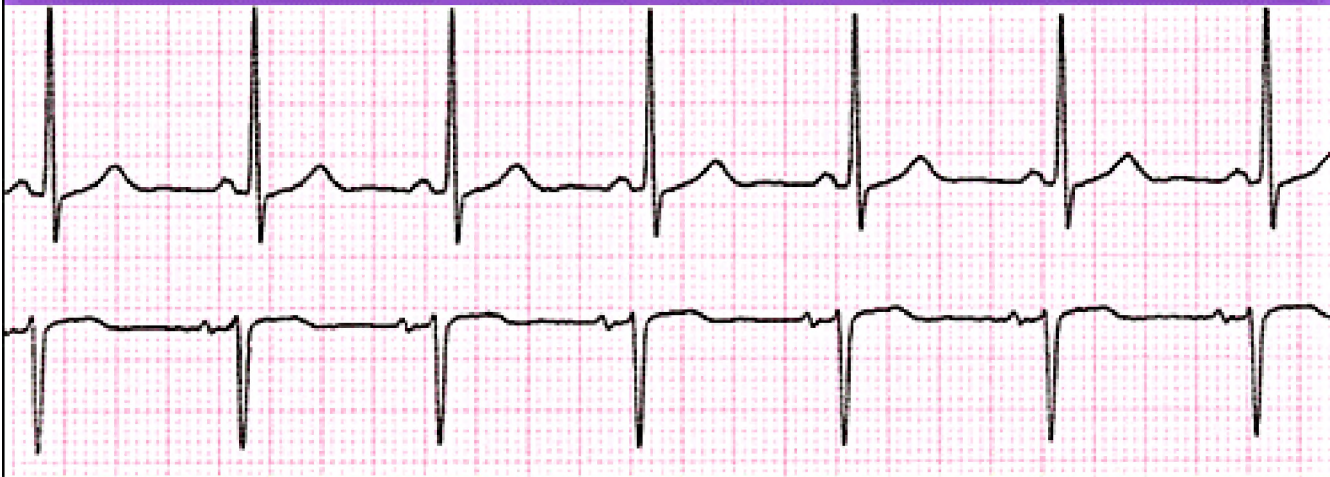


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
<60 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

## Sinus Bradycardia - Marquette-KH

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## Normal Sinus Rhythm



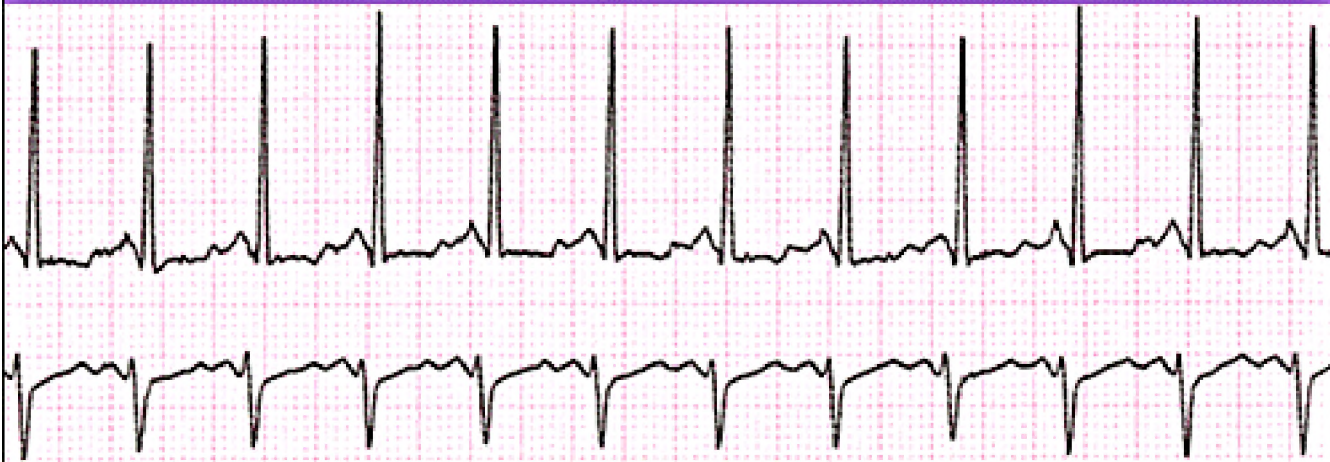
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
60-100 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

## Normal Sinus Rhythm - Marquette-KH

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## Sinus Tachycardia

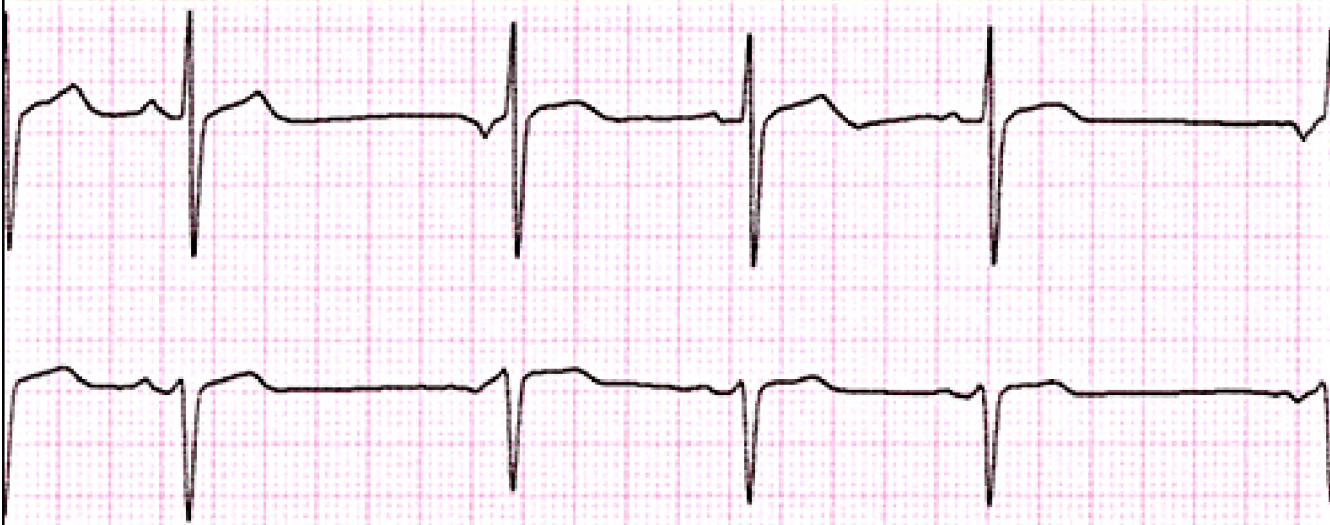


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
> 100 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

## Sinus Tachycardia - Marquette-KH

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## Wandering Pacemaker

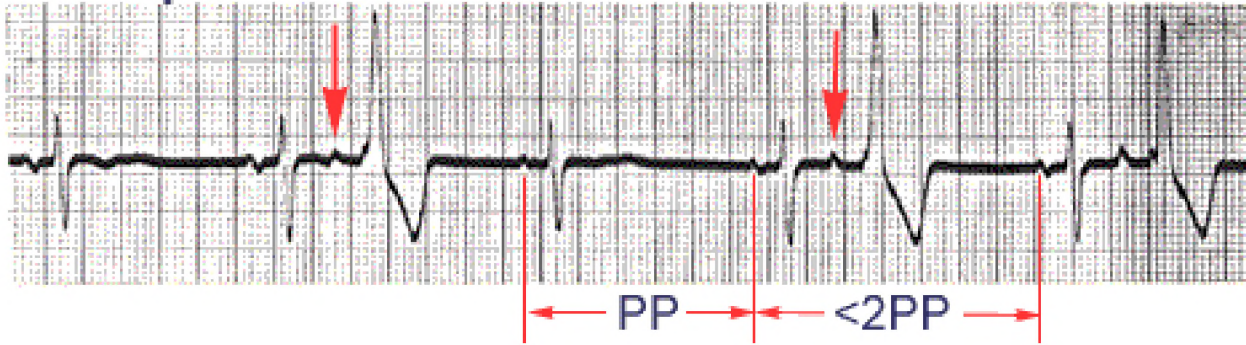


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
Usually <60 bpm	Irregular	Multiple forms	Variable	<.12

## Wandering Atrial Pacemaker - Marquette

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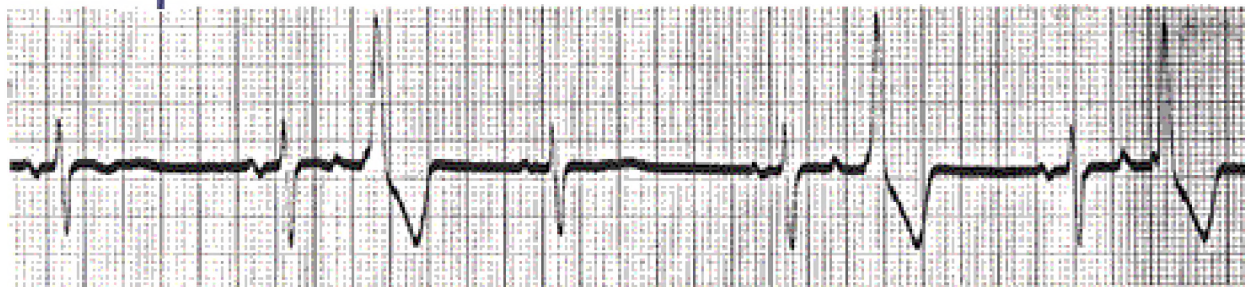
**Lead V<sub>1</sub>**

## PAC's with RBBB Aberrant Conduction

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PAC's are identified by the arrows. Note that the PP interval surrounding the PAC is less than 2x the basic sinus cycle indicating that the sinus node has been reset by the ectopic P wave. The pause after the PAC, therefore, is incomplete.

## Lead V<sub>1</sub>

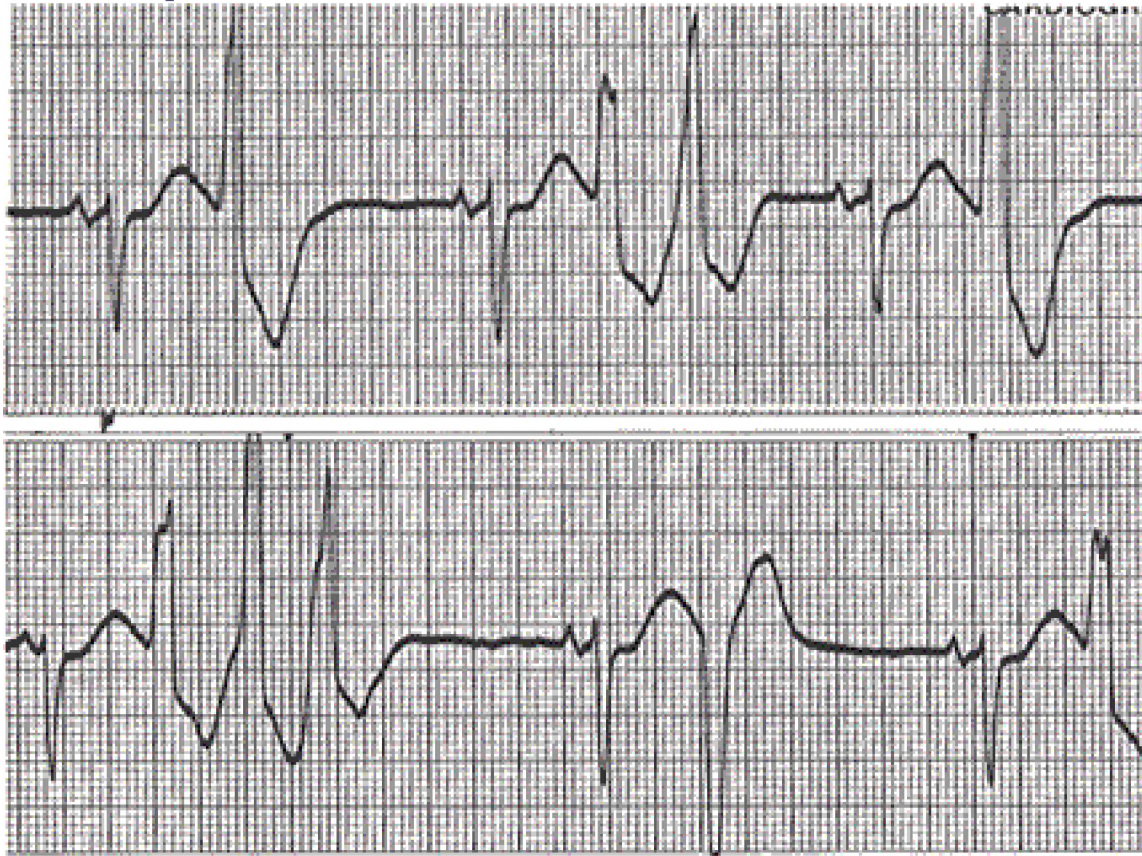


## What are those funny looking beats????

*Frank Yanowitz Copyright 1996*

The differential diagnosis of funny-looking-beats, or FLB's, primarily considers beats of supraventricular origin with aberrant conduction and ventricular ectopic beats. In this example the two FLB's have an easily seen ectopic P wave before them; therefore these are PAC's with RBBB aberration.

## Lead V<sub>1</sub>



**Multifocal PVC's, couplet, triplet in patient with prolonged QT**

## Long QT Mischief

*Frank Yanowitz Copyright 1996*

The long QT ECG has many causes: electrolyte abnormalities including hypo-K, hypo-Mg, and hypo-Ca; drugs including type I antiarrhythmics; CNS injury; and hereditary syndromes. Ventricular arrhythmias are thought to be caused by afterdepolarizations or triggered automaticity.



## Lead V<sub>1</sub>



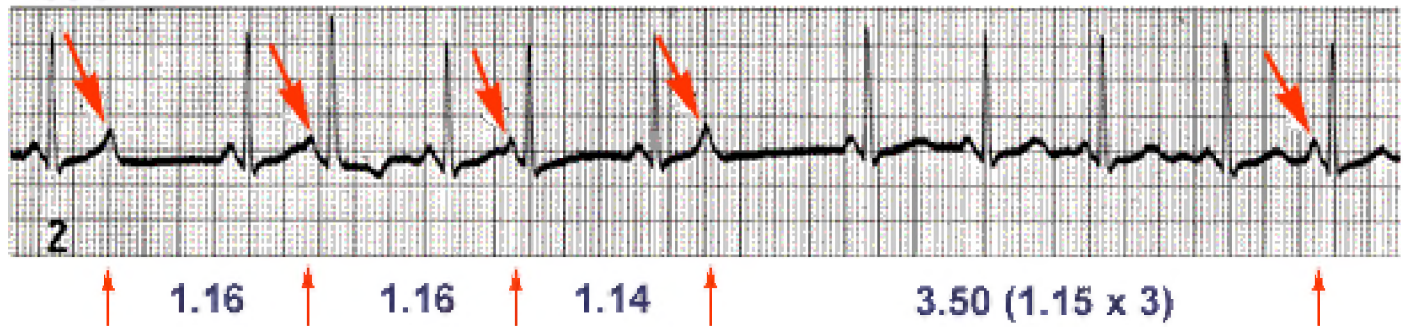
**Atrial fibrillation and PVC's with classic LV morphology**  
**Note position of notch (arrow)**

## Left Ventricular PVC's

*Frank Yanowitz Copyright 1996*

In lead V1, these PVC's are positive or anterior in direction indicating probable LV origin with late activation of the right ventricle. The arrow points to the notch on the downstroke of the PVC making its morphology highly unlikely to be an aberrantly conducted supraventricular beat.

## Lead II



## Atrial Parasystole

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In atrial parasystole non-fixed coupled PACs, shown by arrows, occur at a common inter-ectopic interval or at multiples of this interval. Atrial fusions, not shown here, may also occur when the PAC occurs in close temporal proximity to the sinus impulse.

**Lead V<sub>1</sub>**



**Parasystolic PVC's with fusions (arrows)**

## Ventricular Parasystole

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In ventricular parasystole, non-fixed coupled PVC's occur at a common inter-ectopic interval. Fusion beats, indicated by arrows, are often seen. Fusions occur when the sinus impulse entering the ventricles find the ventricles already partially depolarized by the parasystolic focus.

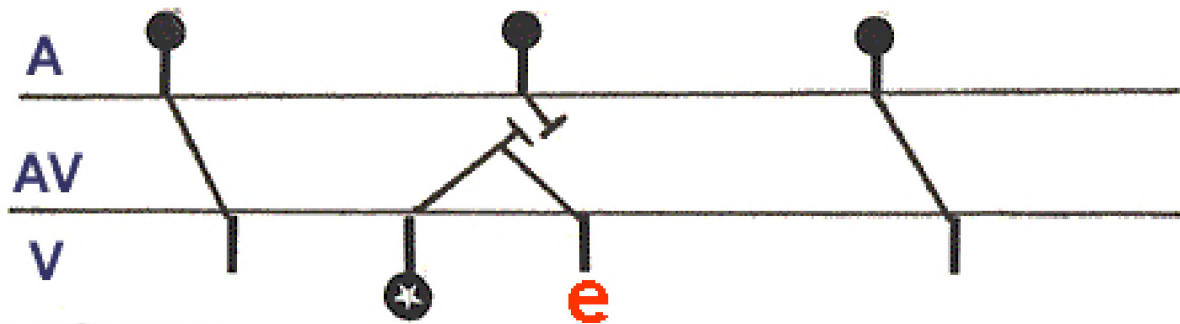
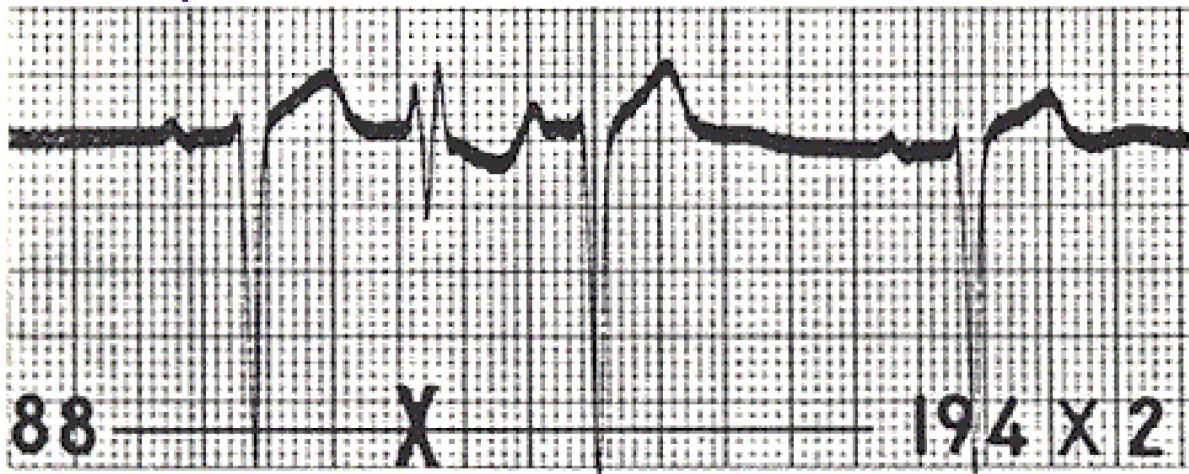


Lead V<sub>1</sub>**A profusion of fusions! (F)**

## Ventricular Fusion Beats

*Frank Yanowitz Copyright 1996*

Fusion beats occur when two or more activation fronts contribute to the electrical event. These may occur in the atria or in the ventricles. In this example the ventricular fusions are the result of simultaneous activation of the ventricles from two foci, the sinus node and a ventricular ectopic focus.

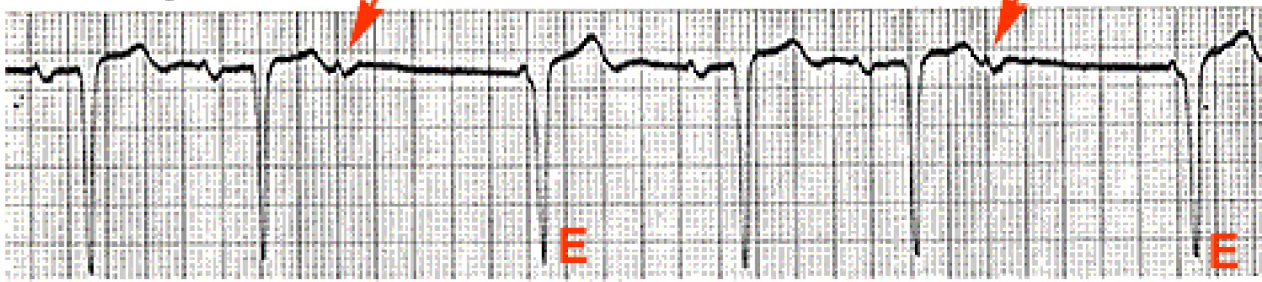
Lead V<sub>1</sub>

Interpolated PVC with ventricular echo (e)

## PVC With Ventricular Echo

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The PVC in this example retrogradely enters the AV junction and returns, usually down a different pathway, to reactivate the ventricles....a ventricular echo. This is unlikely to be an interpolated PVC because the PR interval following the PVC is too short for the sinus impulse to have entered the ventricles.

**Lead V<sub>1</sub>****Nonconducted PAC's (arrows) with junctional escapes (E)**

## Nonconducted PACs and Junctional Escapes

*Frank Yanowitz Copyright 1996*

Although at first glance this looks like 2nd degree AV block, the P waves indicated by the arrows are premature and not sinus P waves. The pause is long enough to encourage a junctional escape focus to take over. Note the sinus P waves just before the escape beats. Had the escapes not occurred, the sinus impulses would have captured the ventricles.



**The most common cause of an unexpected pause!**

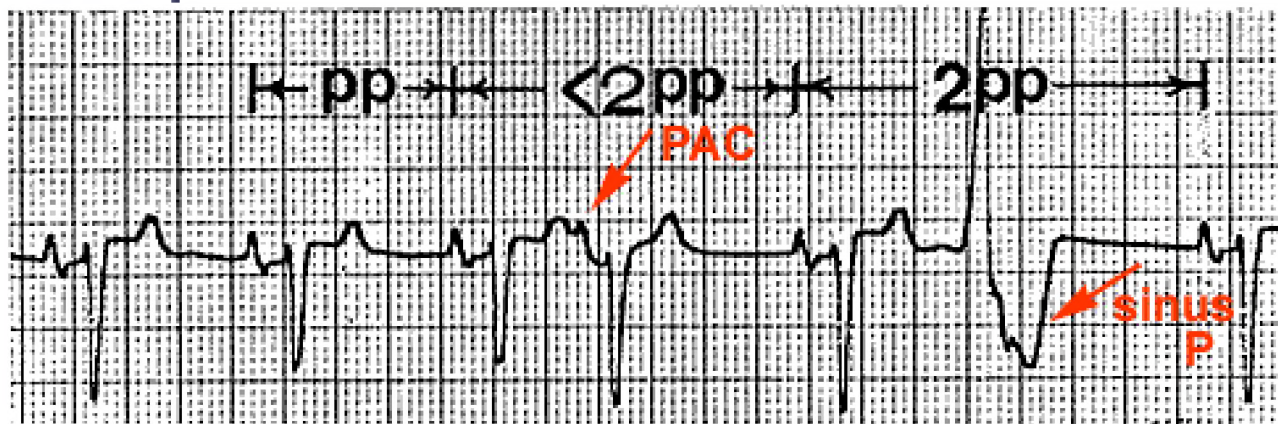
## Nonconducted And Conducted PAC's

*Frank Yanowitz Copyright 1996*

The pause in this example is the result of a nonconducted PAC, as indicated by the first arrow. The second arrow points to a conducted PAC. The most common cause of an unexpected pause in rhythm is a nonconducted PAC.



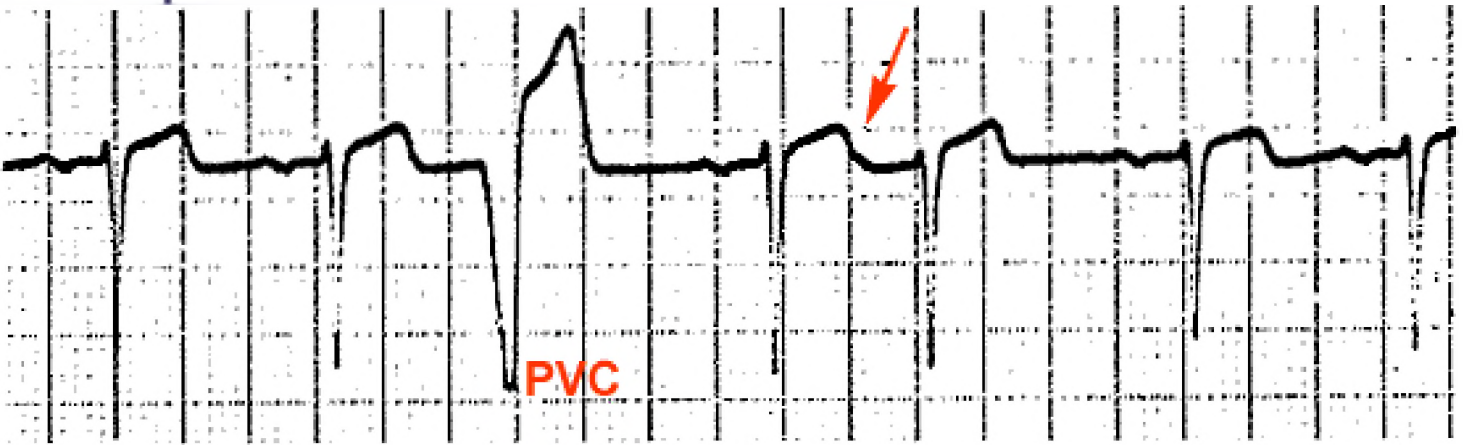
## Lead V<sub>1</sub>



## Complete vs Incomplete Pause

# PAC and PVC: Complete vs. Incomplete Pause-KH

*Frank G. Yanowitz, M.D. copyright 1997*

Lead V<sub>1</sub>

PAC (arrow) - hidden in T-wave

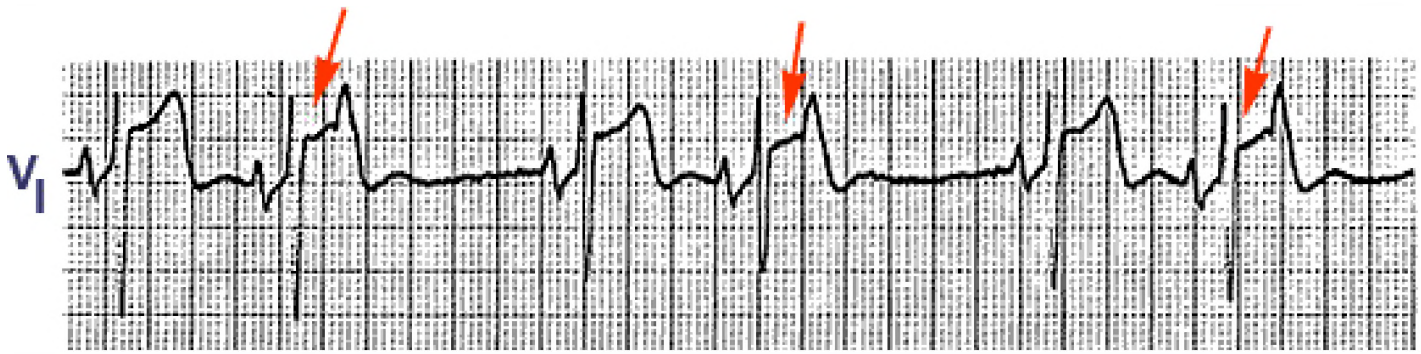
PVC (RV origin) - sticks out like sore thumb

## Identification of PVC's and PAC's-KH

*Frank Yanowitz Copyright 1996*

PVC's usually stick out like sore thumbs; PAC's are often difficult to see because they are hidden in the preceding ST-T wave. The PVC in this example is mostly negative in lead V1 suggesting RV origin; i.e., most of activation is moving in posterior direction towards the left ventricle.



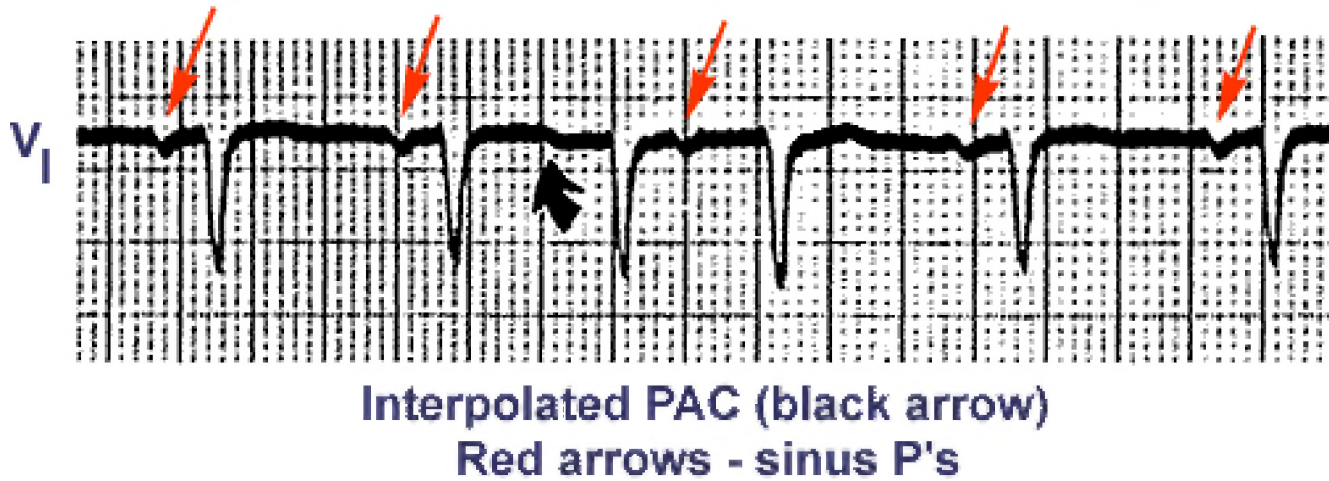


**Nonconducted PAC's (arrows) causing bigeminy**

## **Nonconducted PAC's: An Unusual Bigeminy-KH**

*Frank Yanowitz Copyright 1996*

Occasionally nonconducted PAC's can create interesting rhythms. In this example every other sinus beat is followed by an early, nonconducted PAC. The resulting pause sets up a bigeminal rhythm. Note the distortion of the T waves caused by the nonconducted PAC's.

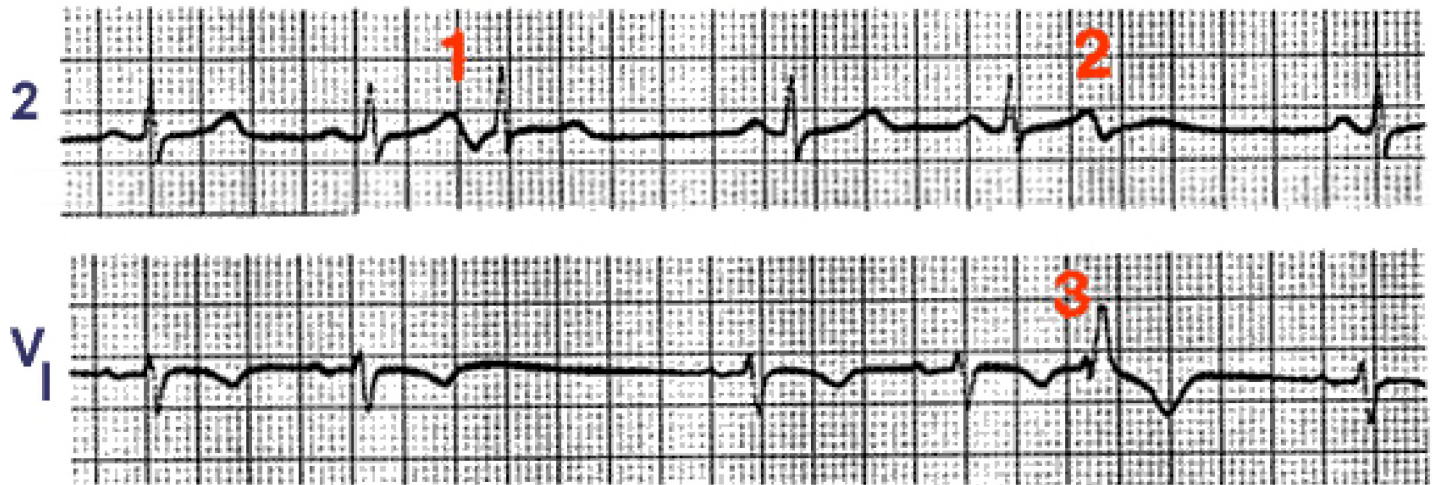


## An Interpolated PAC-KH

*Frank Yanowitz Copyright 1996*

Although most PAC's reset the sinus node producing an "incomplete compensatory pause", this PAC, indicated by the black arrow, is interpolated, i.e., sandwiched between two sinus beats. Note that the subsequent sinus P wave conducts with prolonged PR interval due to the relative refractoriness of the AV junction left by the PAC. Auscultation of the heart during this single PAC event would reveal three rapid beats in a row, suggesting a brief tachycardia.

## Games PAC's play.....

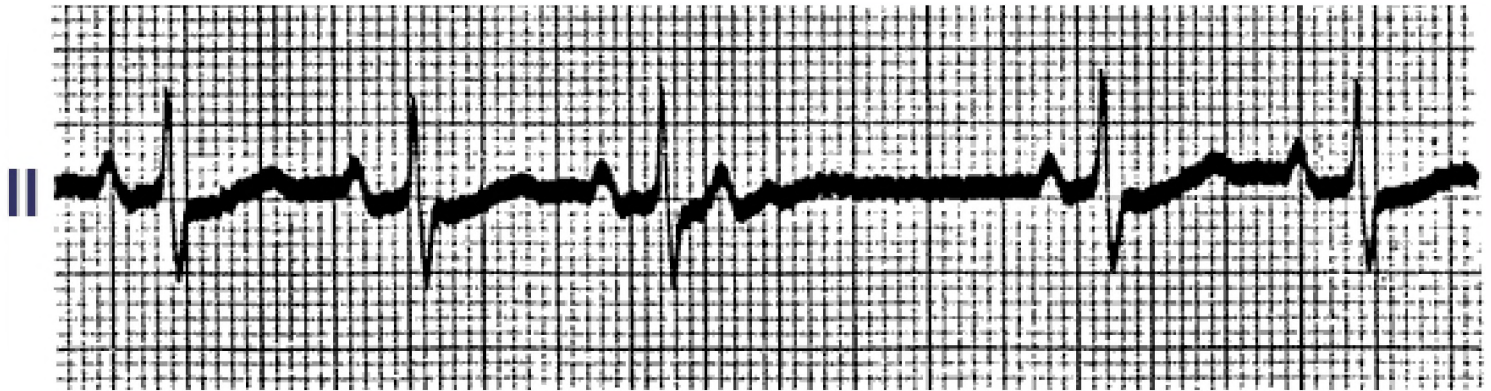


1. PAC with normal IV conduction
2. Nonconducted PAC
3. PAC with RBBB aberration

## The Three Fates Of PAC's-KH

*Frank Yanowitz Copyright 1996*

As illustrated, PAC's can have three fates: PAC-1 enters the ventricles and encounters no conduction delays, therefore causing a narrow QRS; PAC-2 occurs a little earlier and can't get through the AV junction, therefore being "nonconducted"; PAC-3 seen in lead V1 makes it into the ventricles but encounters the right bundle refractory period, therefore conducting with a RBBB morphology; i.e. aberrant conduction.

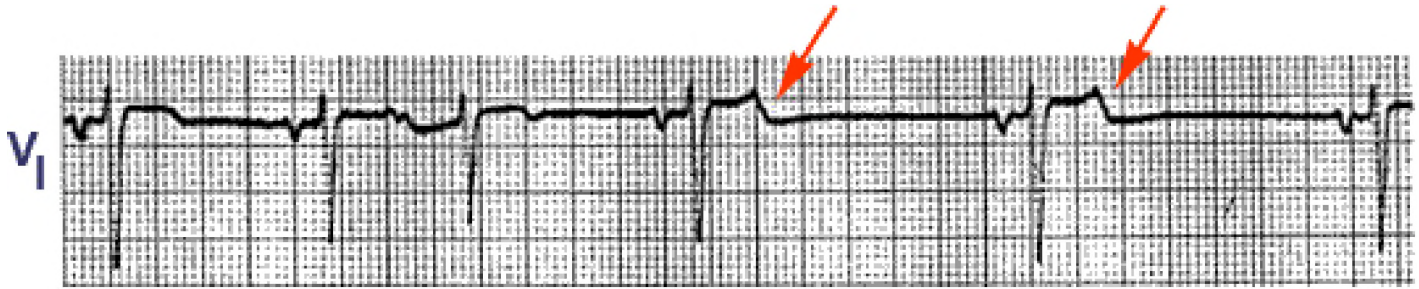


The most common cause of an  
unexpected pause ....

## A Nonconducted PAC Causes An Unexpected Pause-KH

*Frank Yanowitz Copyright 1996*

Unexpected pauses in rhythm have several causes, the most frequent being a nonconducted PAC. In this example the nonconducted PAC is seen in the ST segment of the pause. Note the change in the ST-T compared to the other ST-T waves.



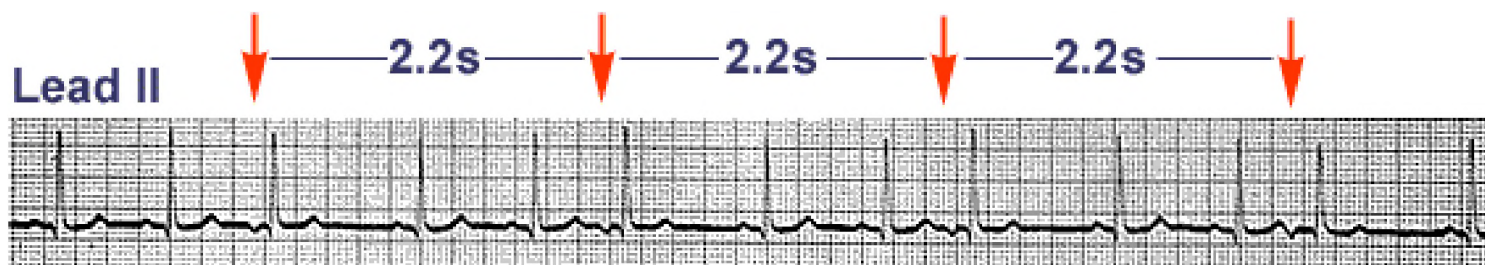
**Nonconducted PAC's (arrows) causing bradycardia**

## Nonconducted PAC's Slowing The Heart Rate-KH

*Frank Yanowitz Copyright 1996*

Consecutive nonconducted PAC's, indicated by arrows, can significantly slow the heart rate. Note the distortion of the ST-T waves caused by the PAC. A hint in recognizing nonconducted PAC's is to find conducted PAC's in the same rhythm strip.





**Atrial parasystole (note common interectopic interval of 2.2s)**

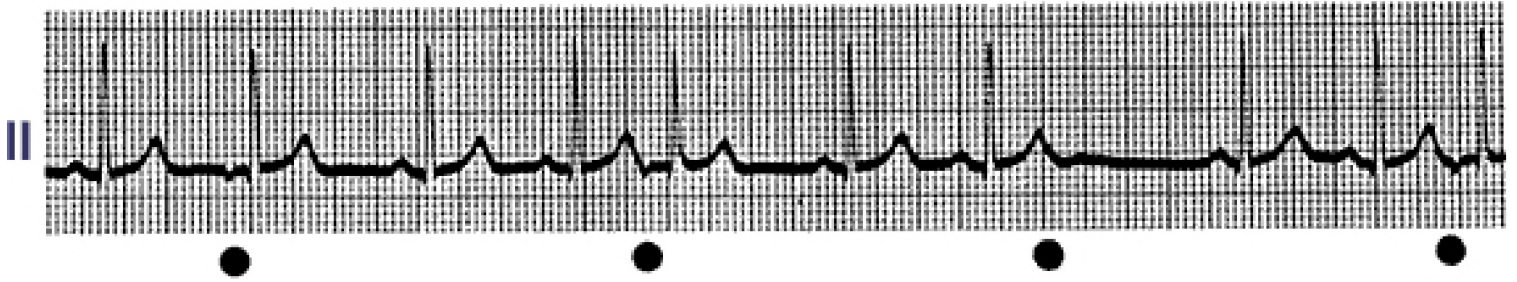
## Atrial Parasystole-KH

*Frank Yanowitz Copyright 1996*

Parasystolic rhythms involve an independent ectopic pacemaker resulting in nonfixed coupled premature beats. Parasystole may occur in the atria, as seen in this example, in the AV junction, and in the ventricles. Note the common inter-ectopic interval separating the parasystolic PAC's.



## What's the rhythm?



## Atrial Parasytostole-KH

*Frank Yanowitz Copyright 1996*

The evenly spaced "dots" indicate ectopic atrial activity from a parasytostolic atrial pacemaker. Non-fixed coupled PAC's are seen having a common inter-ectopic interval. One of the PAC's is nonconducted.

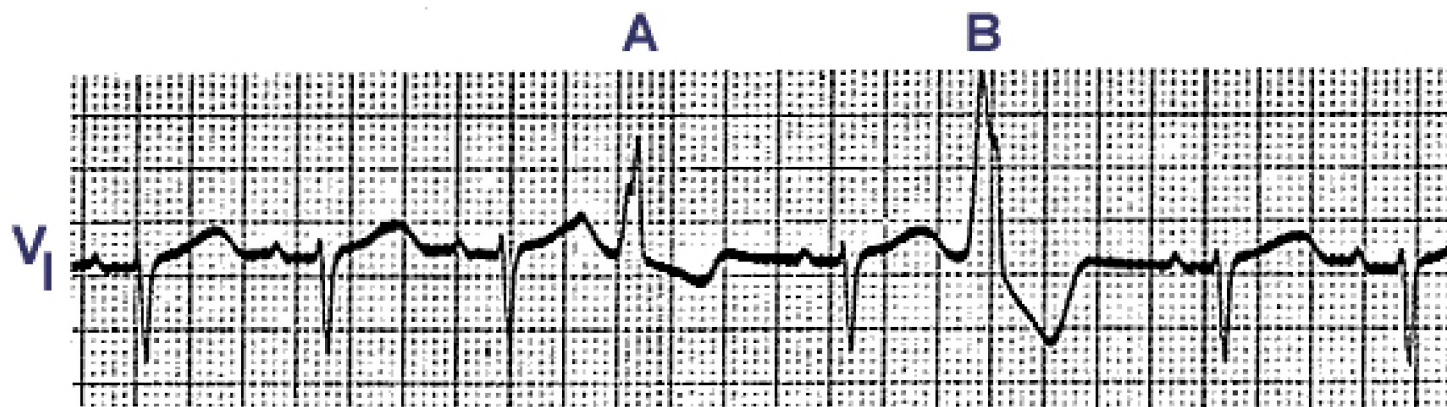
Lead V<sub>1</sub>

**Should patient in 'A' have pacemaker?**

## **Nonconducted and Aberrantly Conducted PAC's-KH**

*Frank Yanowitz Copyright 1996*

In 'A' the slow sinus rhythm is actually caused by nonconducted PAC's hidden in the ST segment. This is confirmed in 'B' where some of the PAC's are aberrantly conducted with LBBB, and some PAC's are nonconducted.

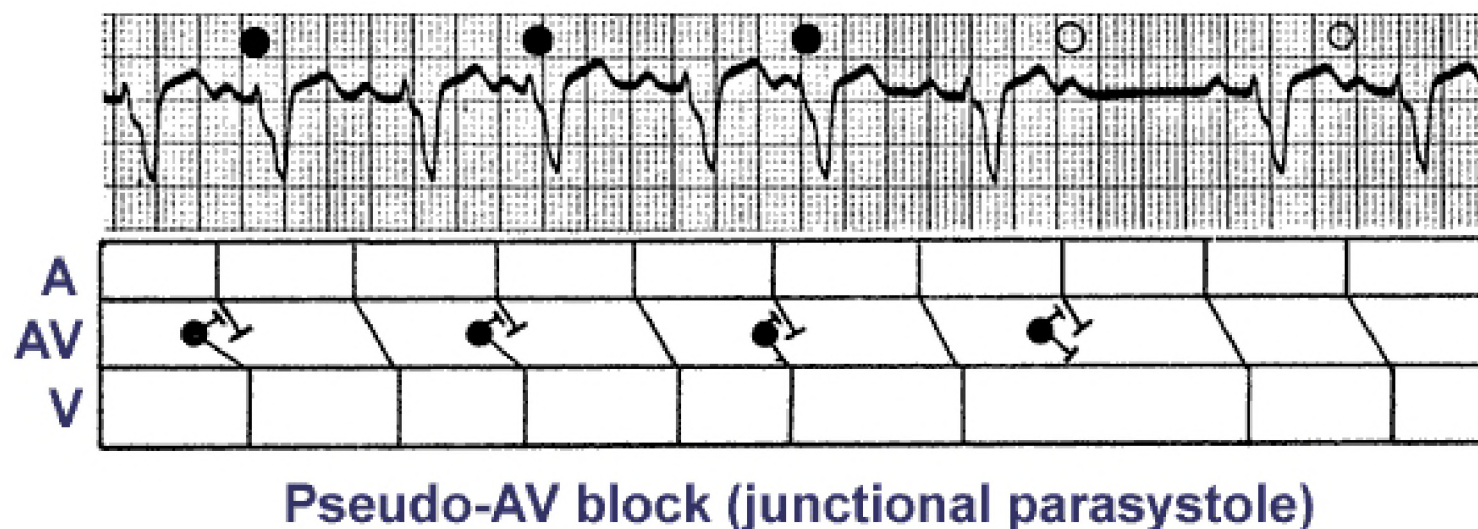


**Can you differentiate sore thumbs 'A' and 'B'?**

## Sore Thumbs-KH

*Frank Yanowitz Copyright 1996*

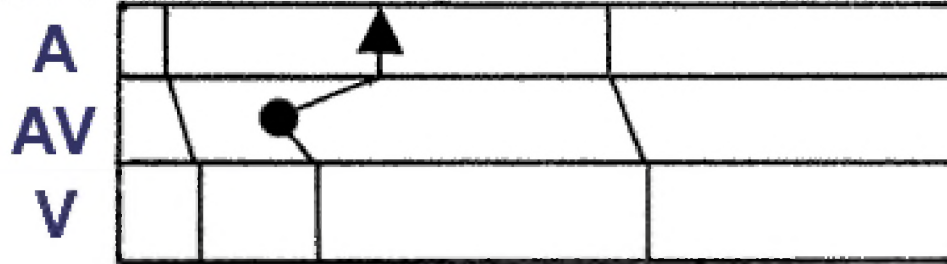
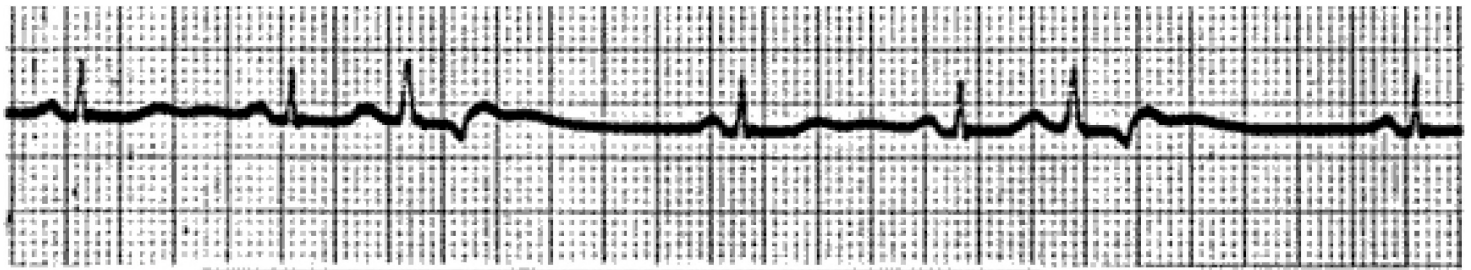
Two funny looking premature beats are seen in this rhythm strip. Beat 'A' is preceded by a PAC which distorts the T wave, making this an aberrantly conducted PAC. Beat 'B' is a PVC. The notch on the downslope of the QRS complex clearly identifies this as a PVC and not aberrancy.



## Junctional Parasystole and Pseudo-AV Block-KH

*Frank Yanowitz Copyright 1996*

This complicated rhythm strip shows normal sinus rhythm and a competing junctional parasystolic focus. Solid circles indicate junctional premature beats from the parasystolic focus. Open circles indicate non-conducted junctional prematures; the first open circle is a nonconducted junctional premature that nevertheless interferes with AV conduction, thus creating the picture of AV block....i.e., pseudo-AV block.



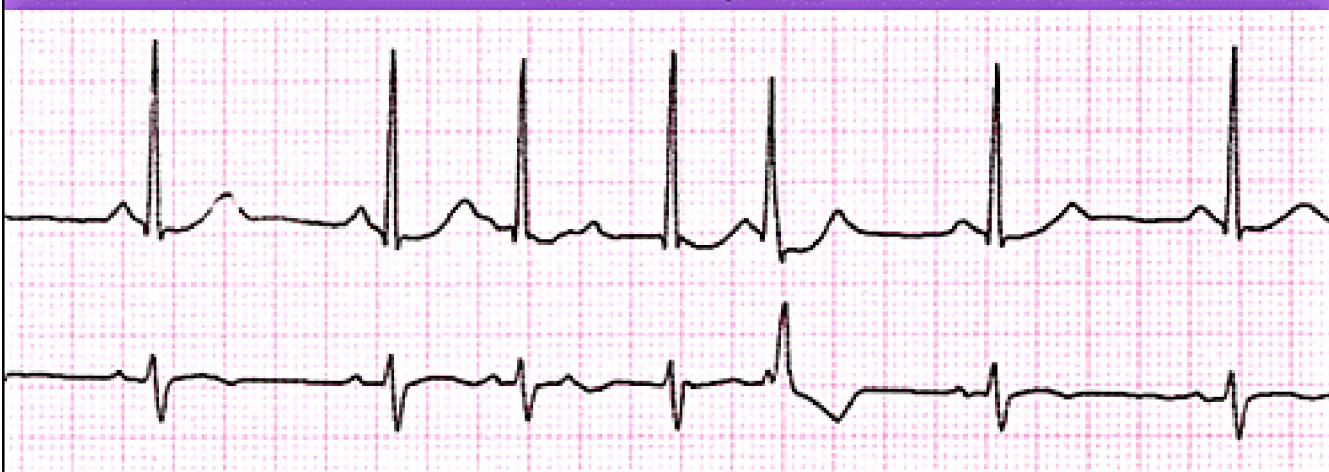
**Premature junctional complexes (PJC's)**

## **Premature Junctional Complexes With Retrograde P Waves-KH**

*Frank Yanowitz Copyright 1996*

The ladder diagram illustrates the PJC with retrograde atrial capture



**Premature Atrial Contraction: Aberrantly Conducted PAC: QRS is Bizarre**

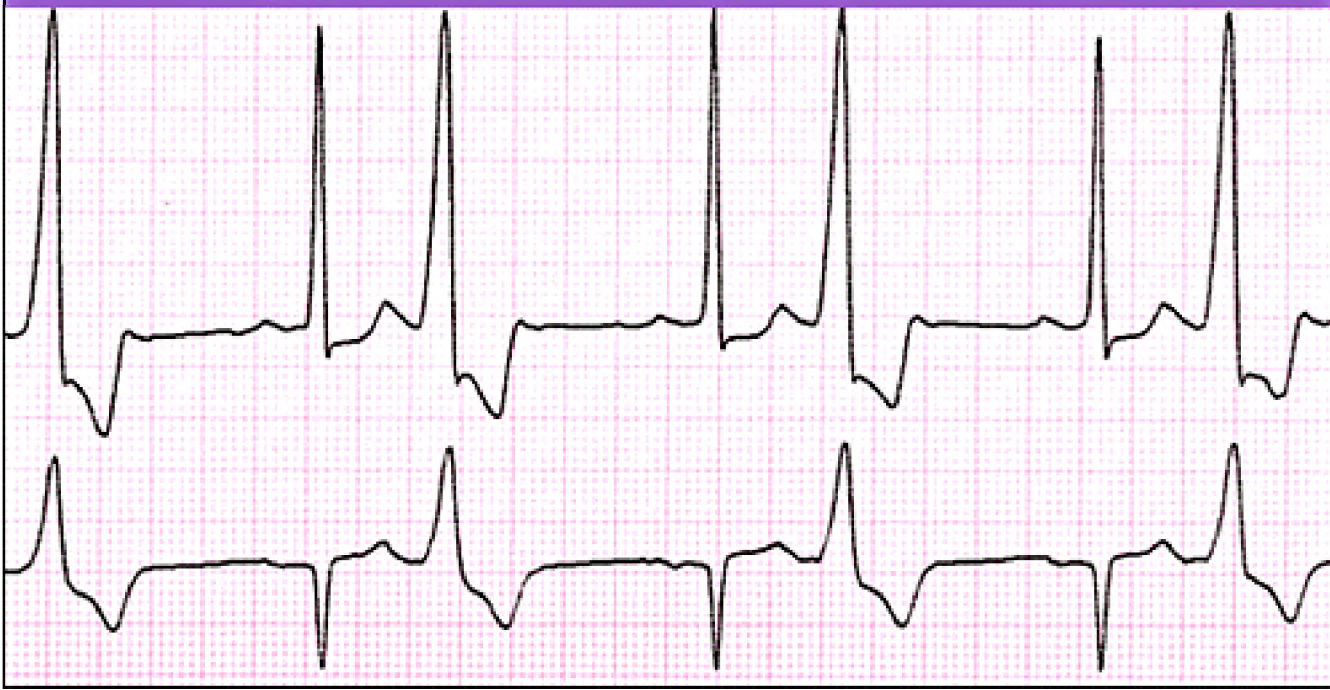
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Premature & abnormal or hidden	<.20	<.12 Abnormal shape

## PAC's With and Without Aberrant Conduction - Marquette-KH

*Marquette Electronics Copyright 1996*

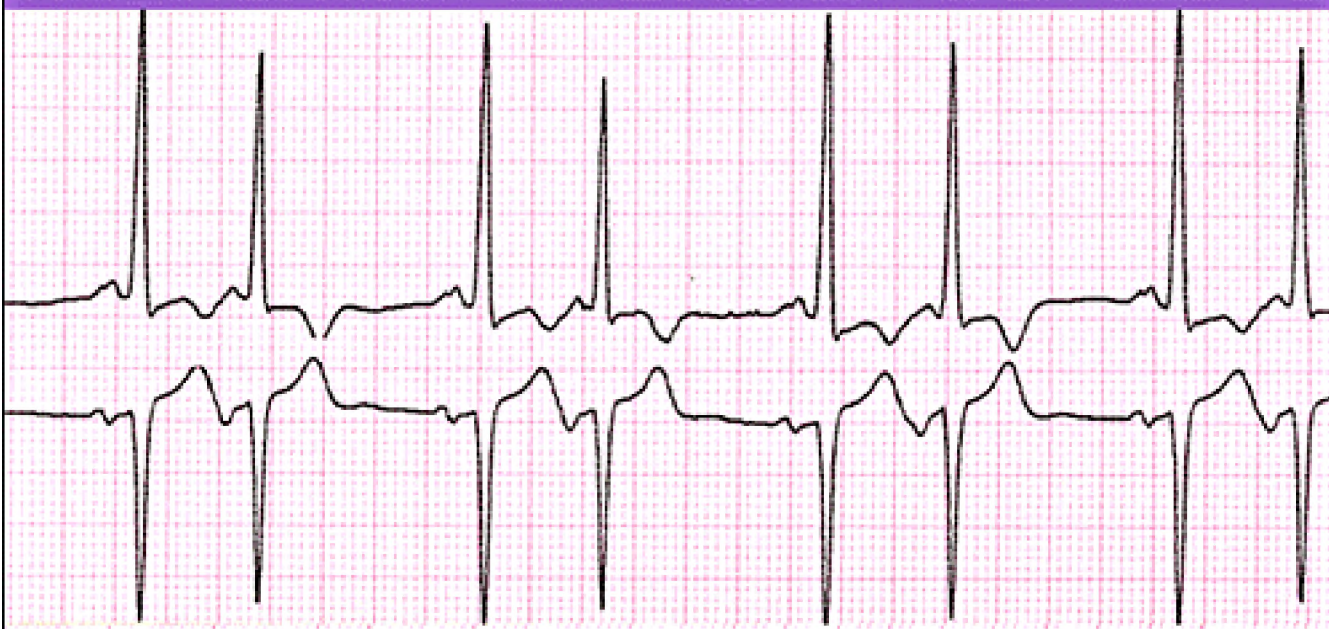


**Bigeminal PVC's: every other beat is a PVC.**



## Ventricular Bigeminy - Marquette-KH

*Marquette Electronics Copyright 1996*

**Premature Atrial Contraction • Atrial Bigeminy: Every Other Beat is a PAC**

Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Premature & abnormal or hidden	<.20	<.12

## Atrial Bigeminy - Marquette-KH

*Marquette Electronics Copyright 1996*

## Coupled PVC's: occur in pairs

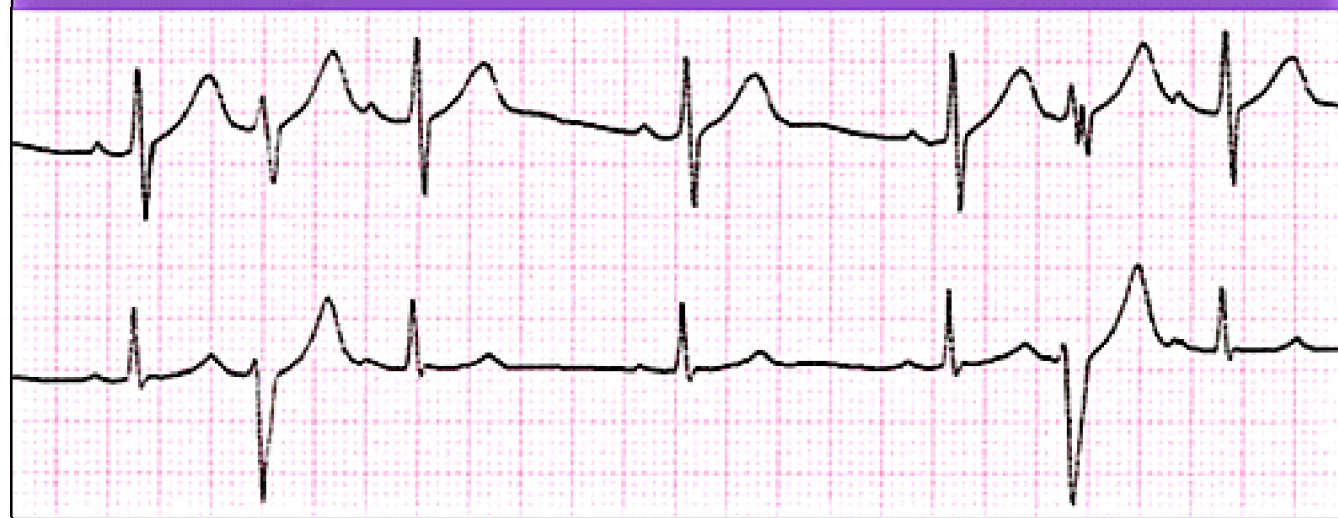


## PVC Couplet - Marquette-KH

*Marquette Electronics Copyright 1996*

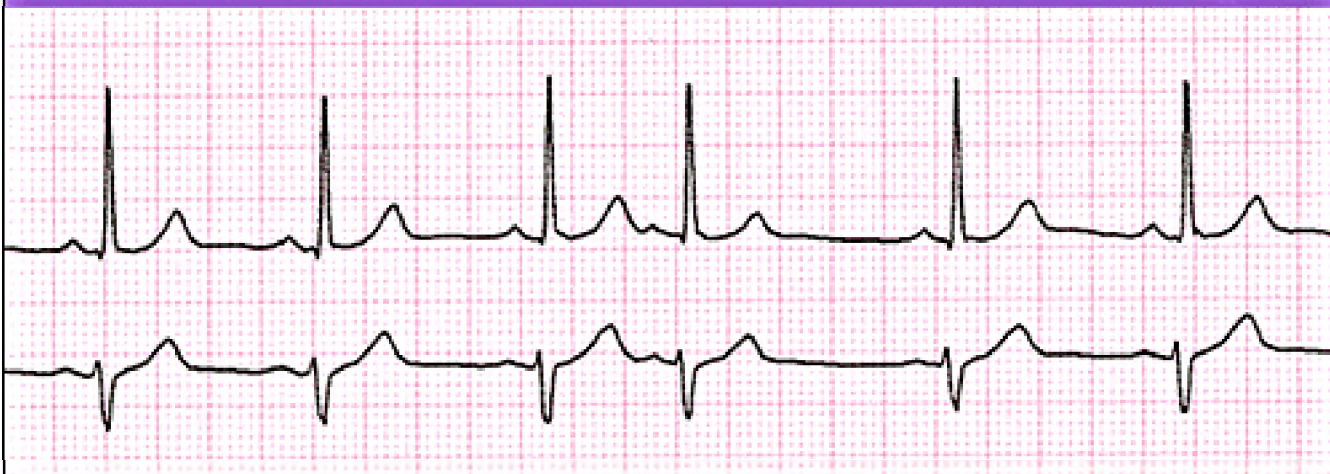


**Interpolated PVC's: occur between sinus beats  
without a compensatory pause**



## Interpolated PVCs - Marquette-KH

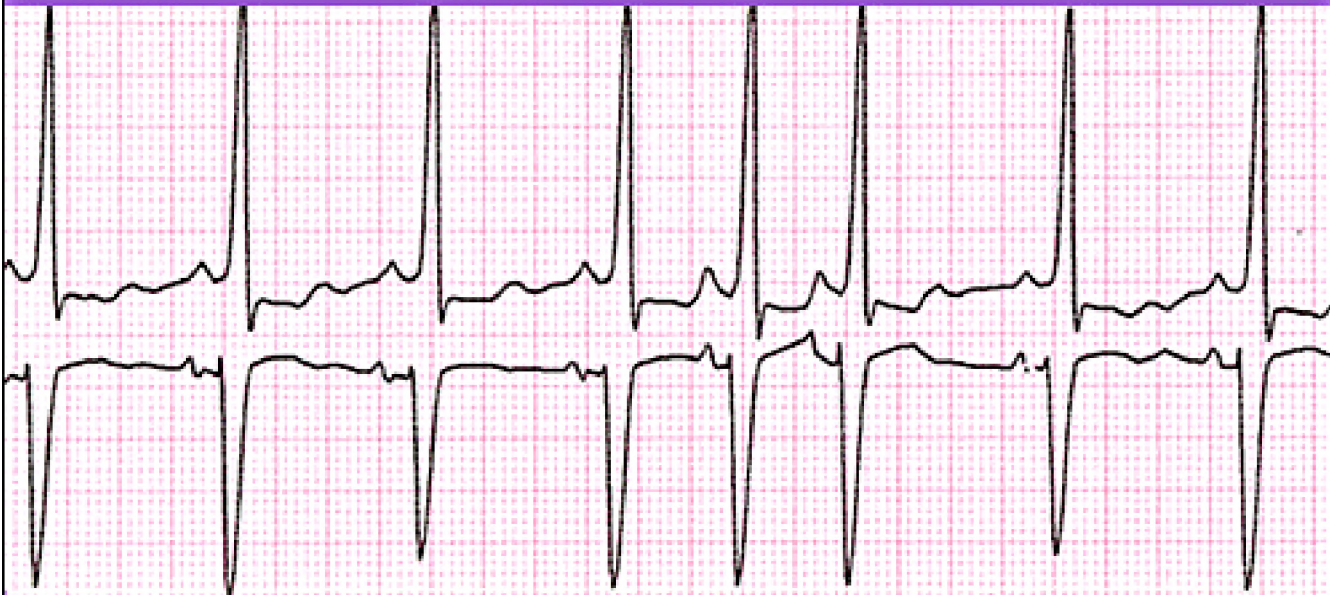
*Marquette Electronics Copyright 1996*

**Premature Atrial Contraction • Isolated PAC's: Occur Single**

Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Premature & abnormal or hidden	<.20	<.12

**Isolated PAC - Marquette-KH**

*Marquette Electronics Copyright 1996*

**Premature Atrial Contraction • Paired PAC's: Occur in Two's****Heart  
Rate**

N/A

**Rhythm**

Irregular

**P Wave**Premature &  
abnormal or  
hidden**PR interval  
(in seconds)**

&lt;.20

**QRS  
(in seconds)**

&lt;.12

## PAC Couplet - Marquette-KH

*Marquette Electronics Copyright 1996*



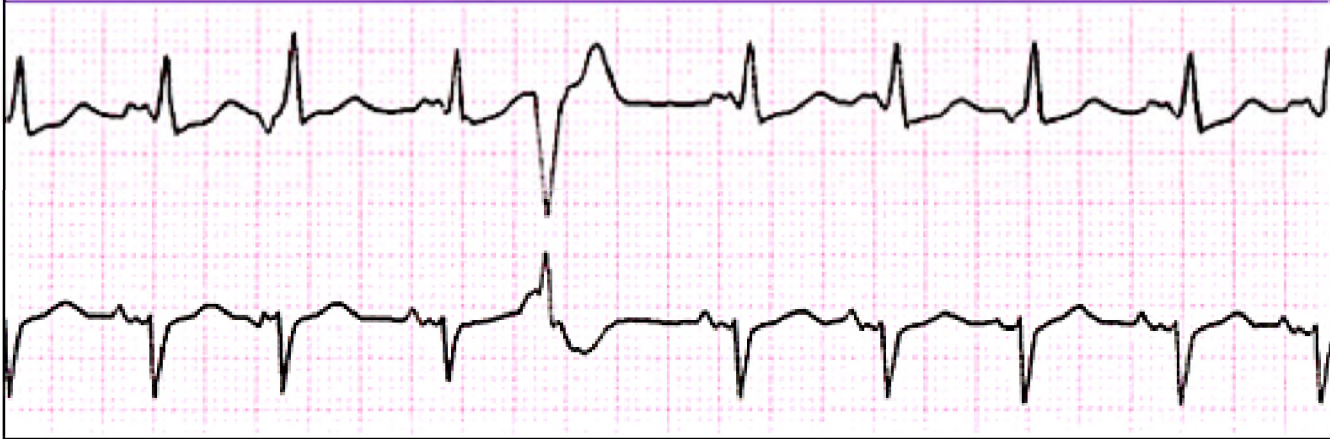
## Quadrigeminal PVC's: every fourth beat is a PVC



## PVC's - Marquette-KH

*Marquette Electronics Copyright 1996*

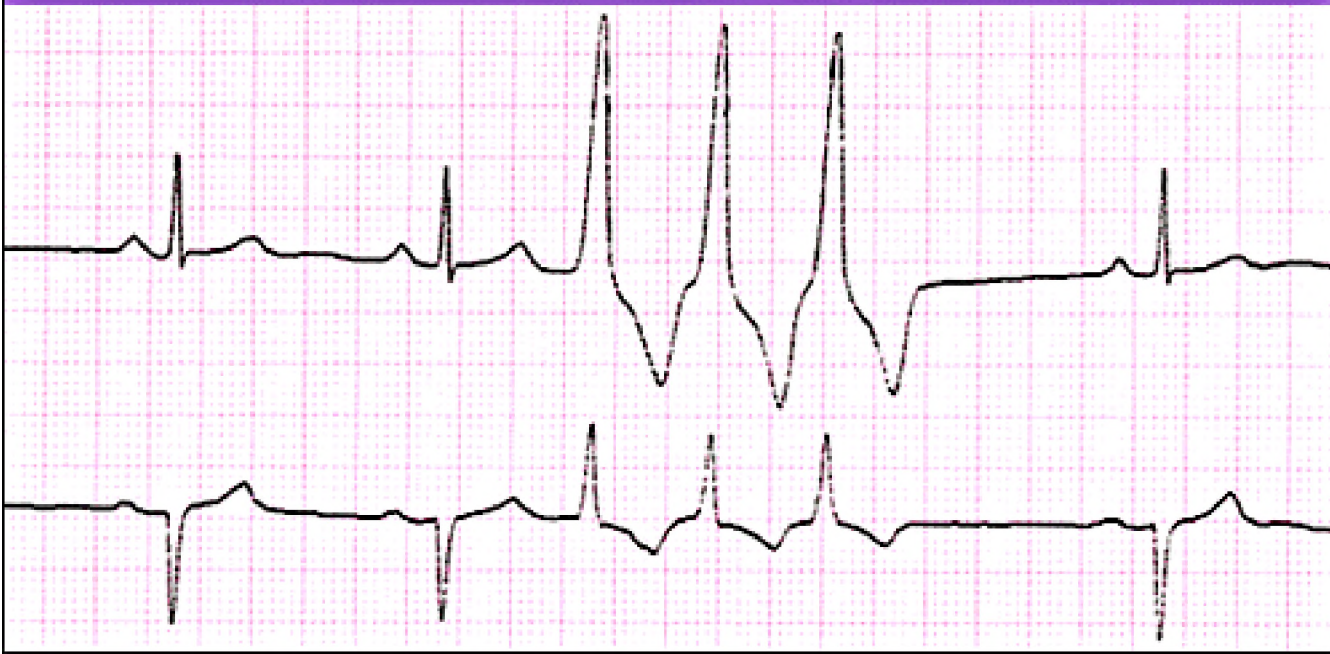
**R on T: occur on the peak of the T wave of the preceding beat**



## PVC with R-on-T - Marquette-KH

*Marquette Electronics Copyright 1996*

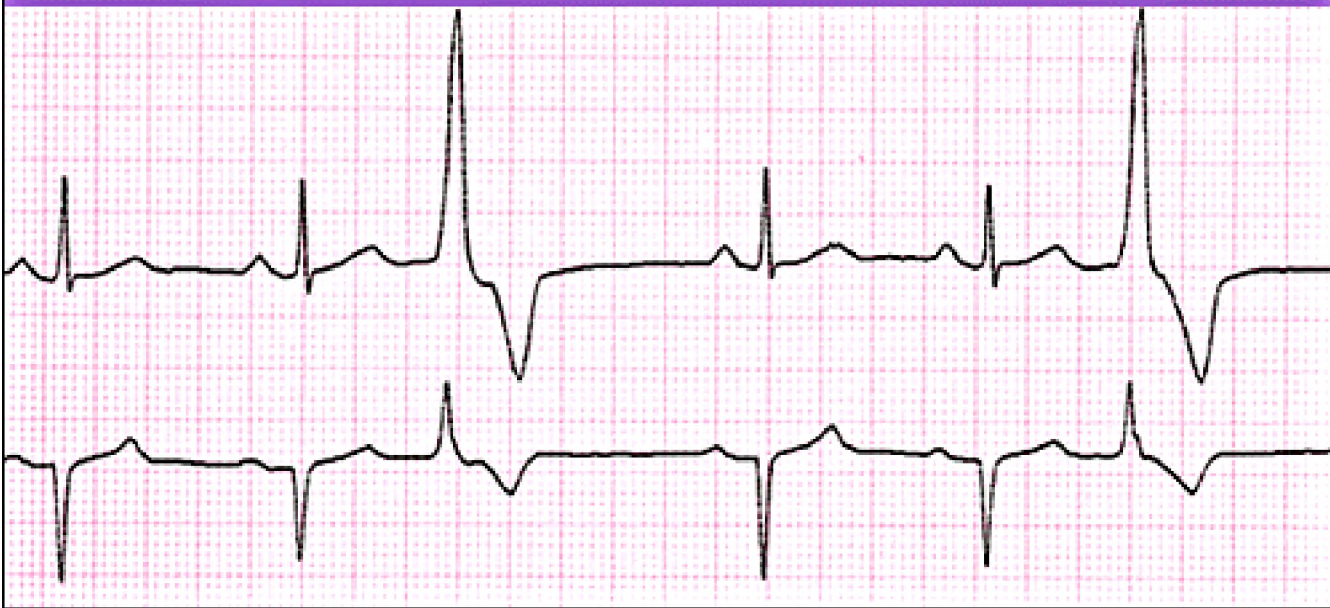
## Triplet PVC's: occur in groups of three



## PVC Triplet - Marquette-KH

*Marquette Electronics Copyright 1996*

## Trigeminal PVC's: every third beat is a PVC

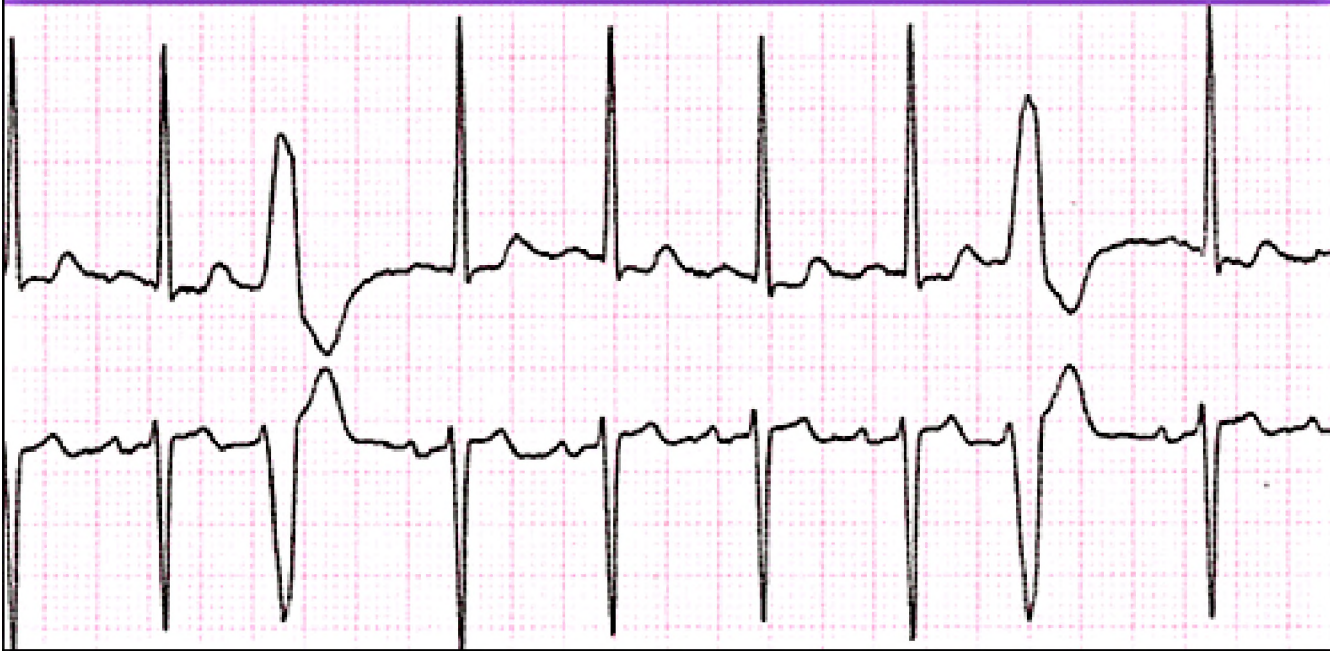


## PVCs - Marquette-KH

*Marquette Electronics Copyright 1996*

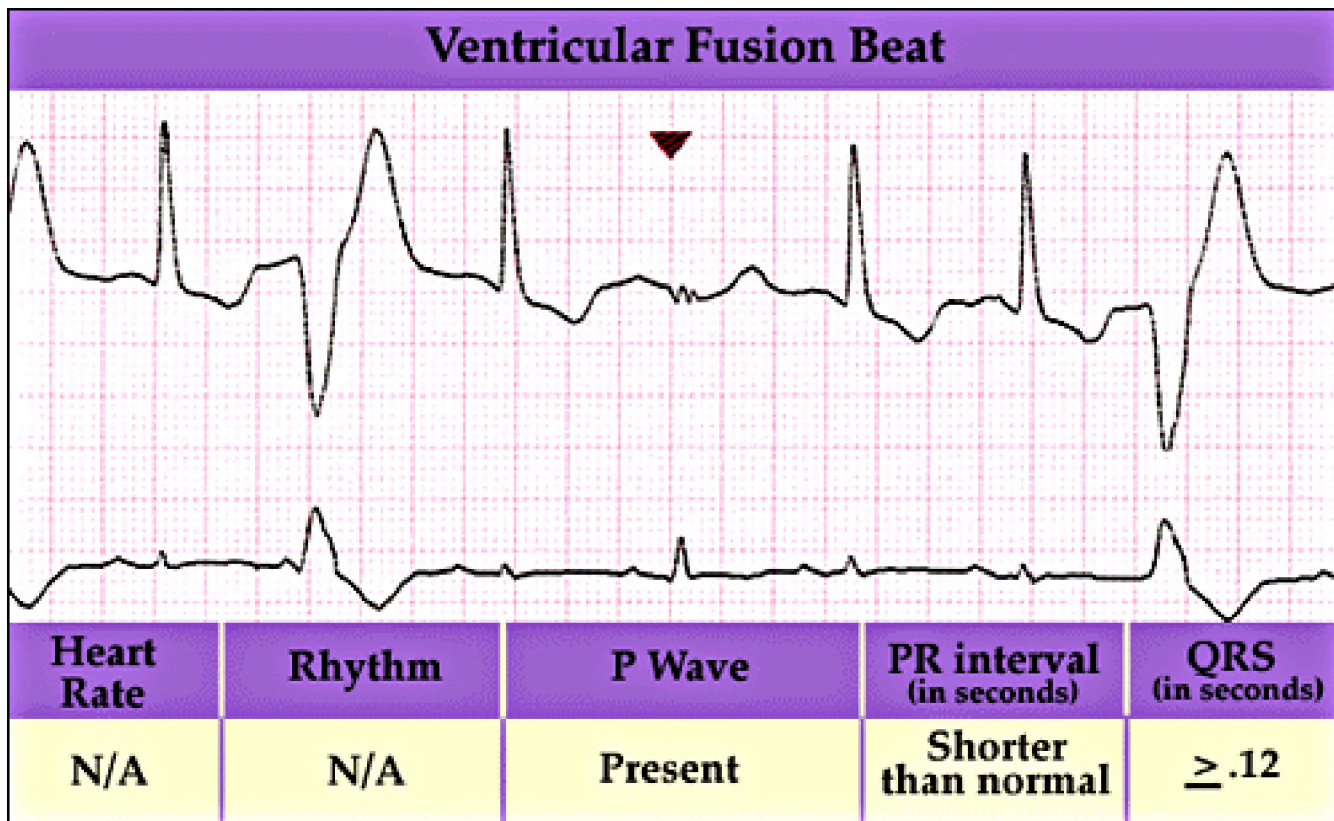


**Unifocal PVC's: identical shapes**  
**Note: A single PVC is labeled isolated**



## Unifocal PVCs - Marquette-KH

*Marquette Electronics Copyright 1996*

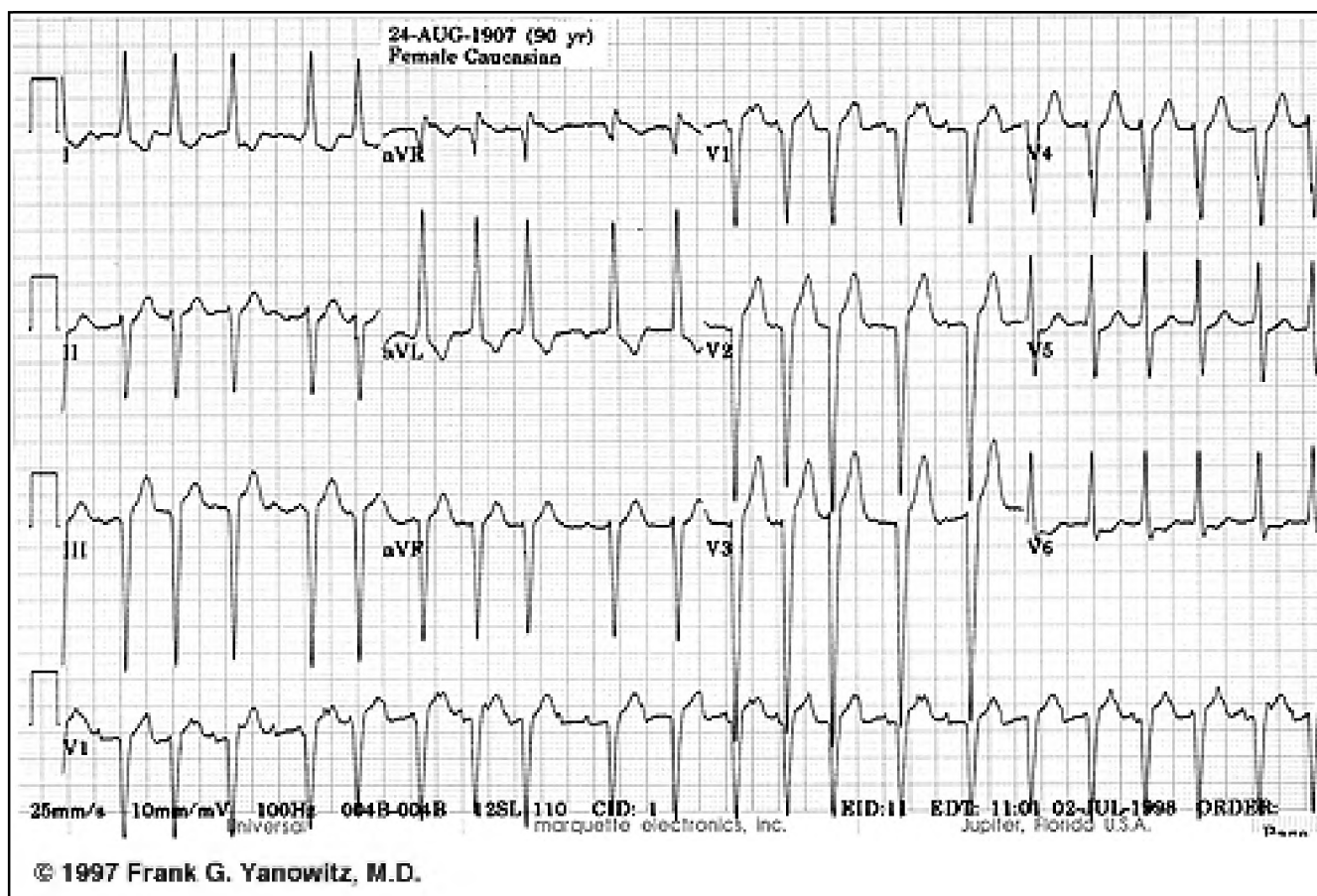


## Ventricular Fusion Beat - Marquette-KH

*Marquette Electronics Copyright 1996*

A ventricular fusion beat represents the simultaneous activation of the ventricles by two independent wavefronts. In this example one wavefront originates in the PVC focus, and the other is from the sinus node. Note the presence of the P wave before the fusion. The QRS of the fusion looks a bit like the PVC and a bit like the sinus QRS.

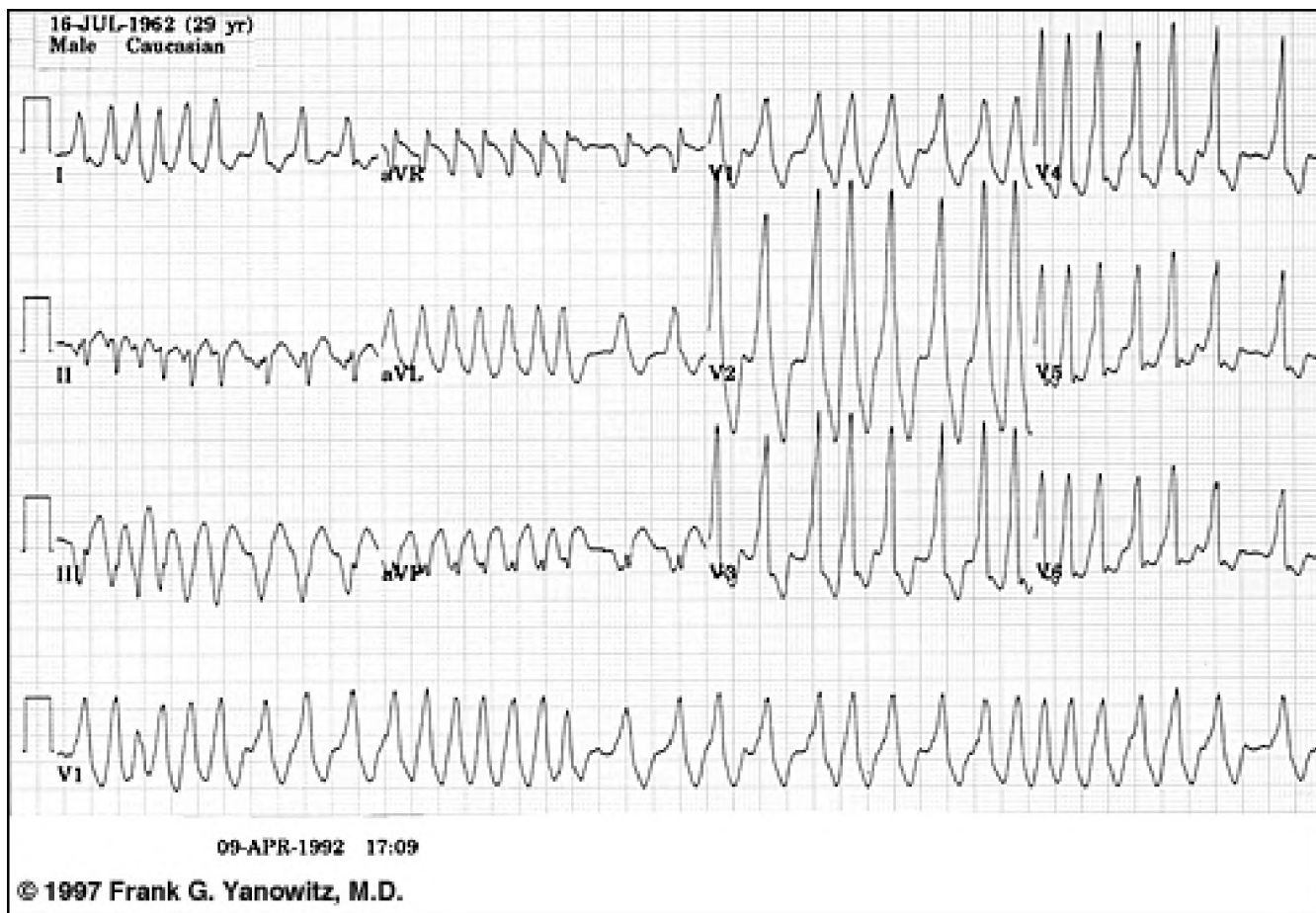




## Multifocal Atrial Tachycardia (MAT)

*Frank G. Yanowitz, M.D. Copyright 1998*

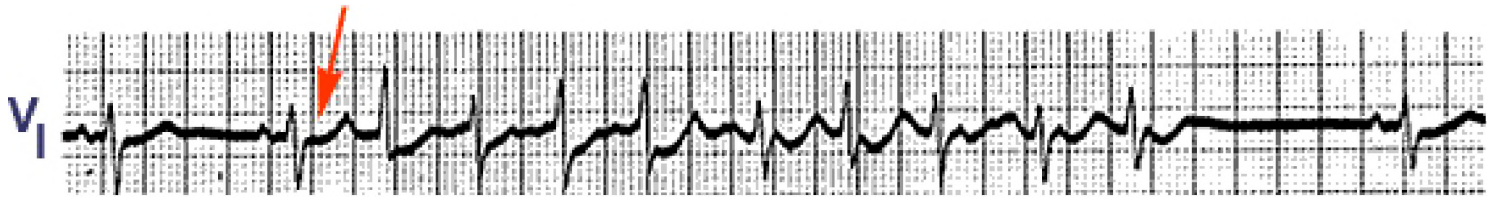
The features of MAT are best seen in the long V1 rhythm strip. P waves of at least 3 different morphologies are present. The ventricular rate is "irregularly irregular" with the main differential diagnosis being atrial fibrillation. In many of the leads, this ECG looks just like atrial fib. Also present are marked left axis deviation, probably due to left anterior fascicular block, and diffuse ST-T wave abnormalities.



## Atrial Fibrillation in Patient with WPW Syndrome

*Frank G. Yanowitz, M.D. Copyright 1998*

This bizarre wide QRS tachycardia is "irregularly irregular", indicative of atrial fibrillation with a fast ventricular response. The bizarre QRS morphology is due to ventricular activation being initiated from the AV bypass track (bundle of Kent) which conducts faster than the AV node.



**Brief atrial fibrillation initiated by early PAC (arrow)**

## **A PAC Initiates Paroxysmal Atrial Fibrillation-KH**

*Frank Yanowitz Copyright 1996*

The arrow indicates slight alteration of the ST-T wave by a PAC. The PAC, in turn, falls during the vulnerable period of atrial repolarization and initiates atrial fibrillation. Similar but more catastrophic events happen in the ventricles when PVC's occur during the vulnerable period, i.e. R-on-T, of ventricular repolarization

## Lead V<sub>1</sub>



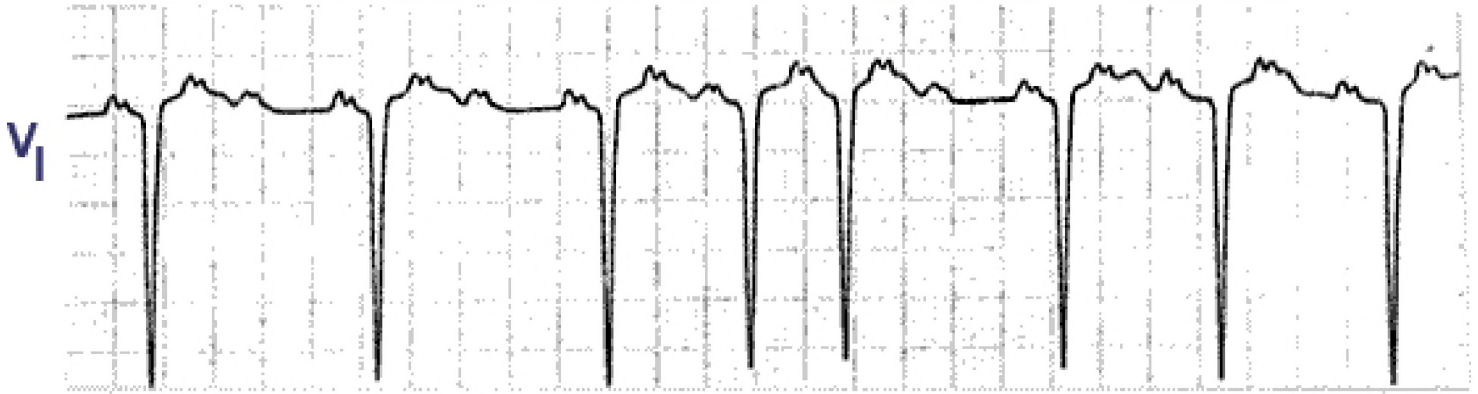
## Atrial tachycardia with variable AV block

# Atrial tachycardia With 3:2 AV Block-KH

*Frank Yanowitz Copyright 1996*

In this rhythm the atrial rate from an ectopic focus is 160 bpm. Atrial activity can be seen on top of T waves, and before QRS's. Careful observation reveals a 3:2 Wenckebach relationship between P waves and QRS's. Atrial tachycardia with block is often a sign of digitalis intoxication.

## Digitalis intoxication (2 levels of block)



1. Atrial tachycardia with exit block (note group beating in atria)
2. AV block

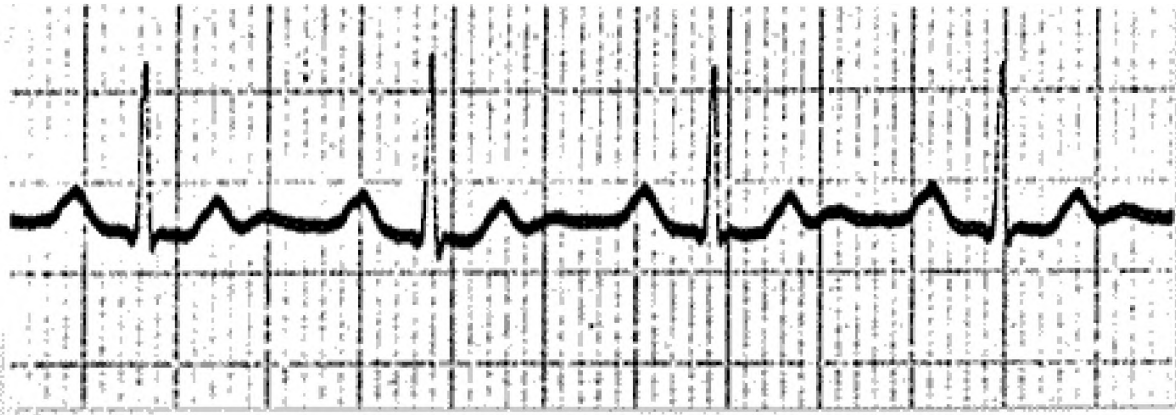
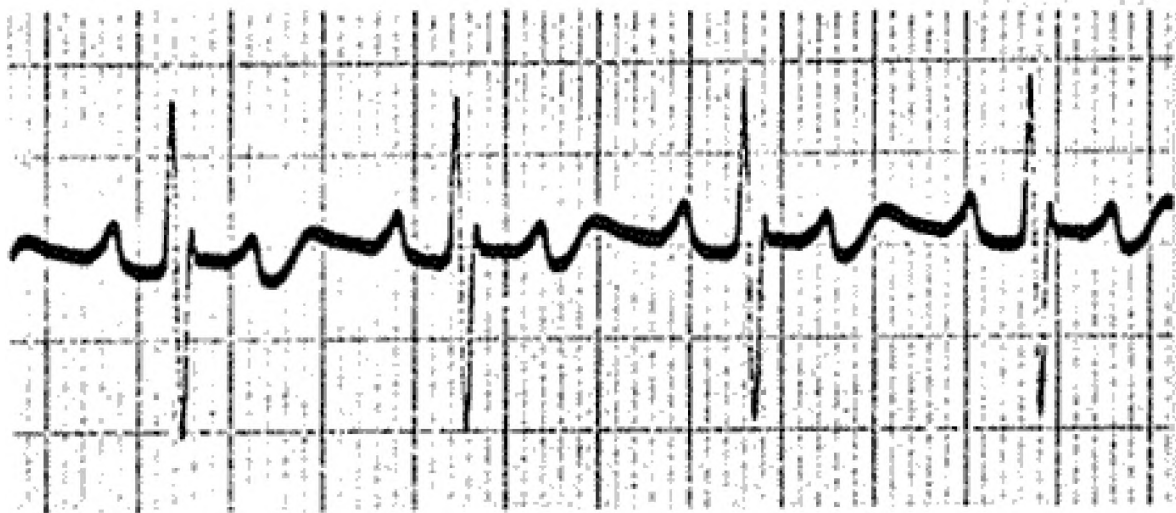
## Atrial Tachycardia With Exit Block and AV Block-KH

*Frank Yanowitz Copyright 1996*

The ectopic P waves, easily seen in this example, occur in groups, separated by short pauses. This is likely due to an exit block just distal to the atrial pacemaker. Because not all of the P waves make it to the ventricles, there is also 2nd degree AV block. Therefore, two levels of block are present: one in the atria and one at the level of the AV junction.



## Diagnosis?

**II****aVL****V<sub>I</sub>**

**Middle-aged woman with GI symptoms.  
Medications unknown.**

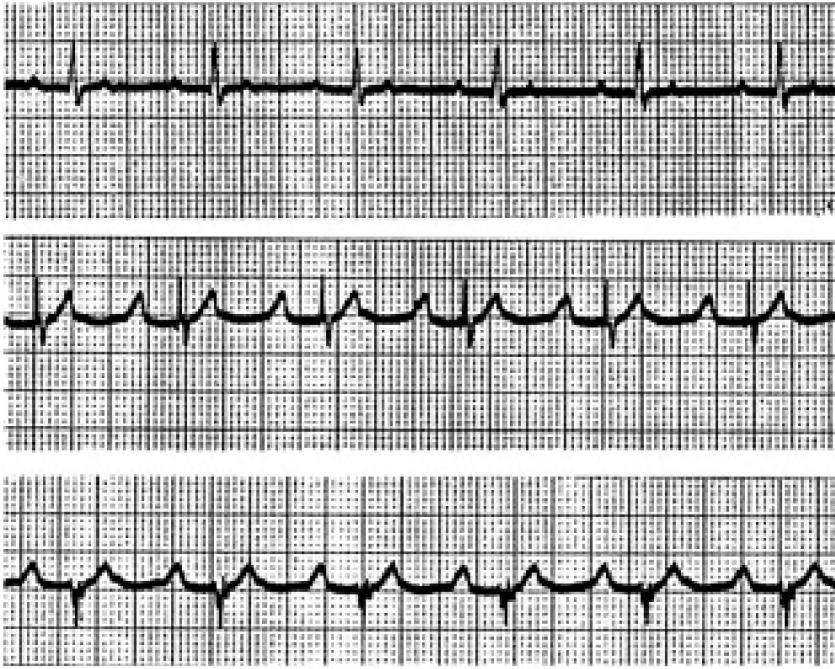
**A Very Subtle Atrial Tachycardia With 2:1 Block-KH**



*Frank Yanowitz Copyright 1996*

Although at first glance this looks like normal sinus rhythm at 95 bpm. On closer look, there are 2 'P' waves for every QRS; the atrial rate is 190 bpm. Note the hidden 'P' in the T waves. This rhythm is likely due to digitalis intoxication, as are the GI symptoms.

## Digitalis Toxicity

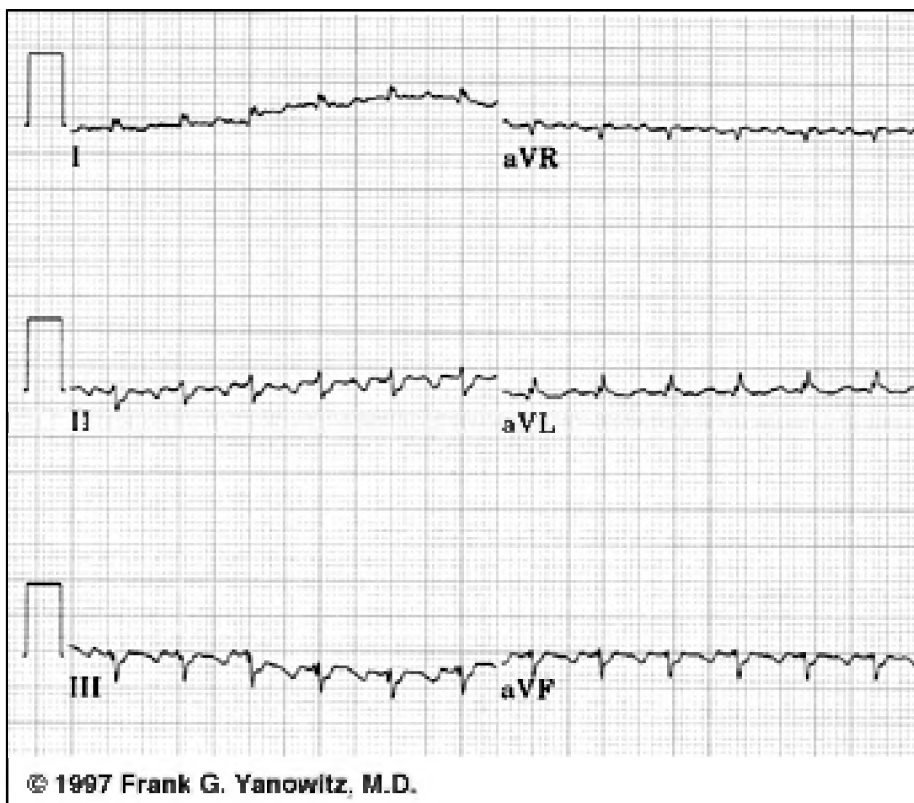


Atrial tachycardia, 2:1 AV block

1. The P-wave axis is normal
2. The non-conducted P-wave hides in the T-wave
3. The conducted P-wave often has a long PR interval
4. The P-P interval may not be exactly regular

# Atrial Tachycardia With 2:1 AV Block: A Manifestation of Digitalis Intoxication-KH

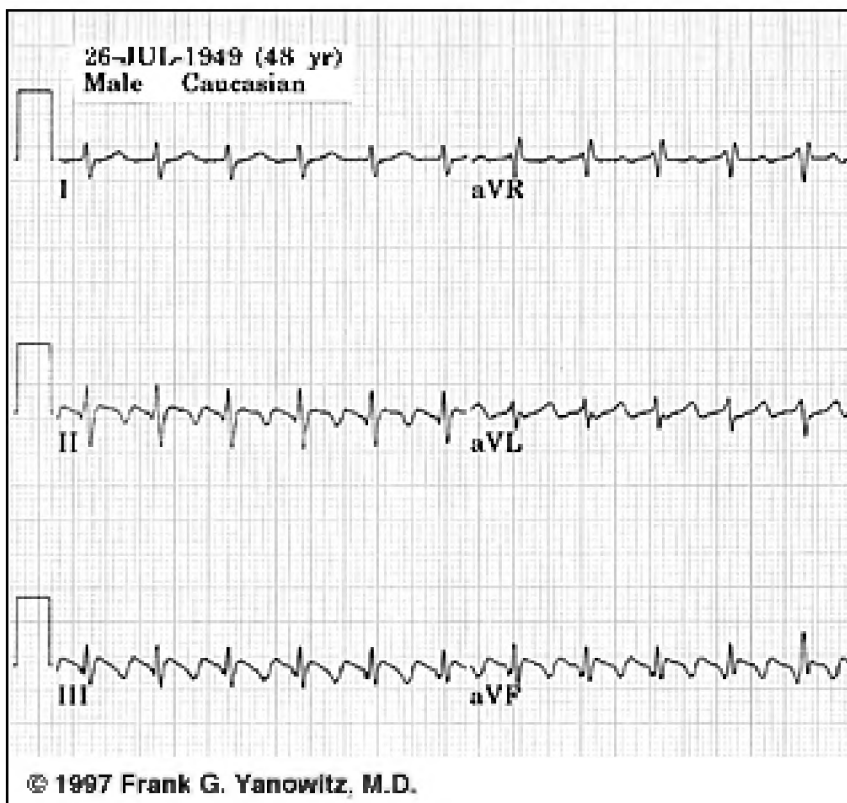
*Frank Yanowitz Copyright 1996*



## Atrial Flutter with 2:1 AV block

*Frank G. Yanowitz, M.D. Copyright 1998*

Whenever there is a supraventricular tachycardia with a regular rate of around 150 bpm, THINK "flutter with 2:1 block" before considering anything else. In this 6-lead ECG the flutter waves are best seen in leads II, III, aVF, but one of the flutter waves is at the tail end of the QRS complex, making the QRS appear wider than it actually is.

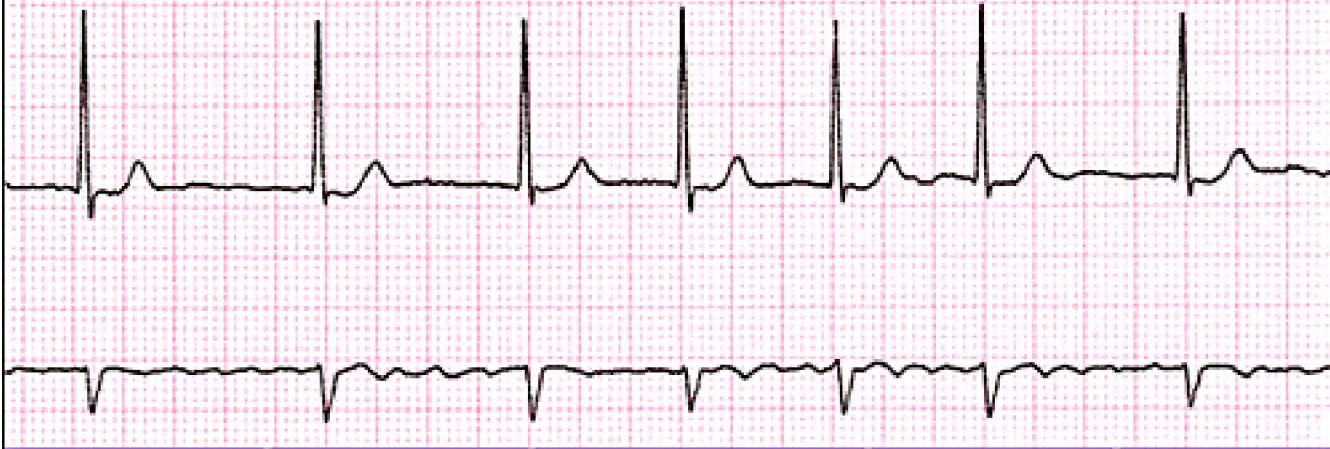


## Atrial Flutter with 2:1 AV Block

*Frank G. Yanowitz, M.D. Copyright 1998*

Atrial flutter with 2:1 AV block is the most frequently missed regular supraventricular tachycardia. In this 6 lead ECG the heart rate is 150 bpm which should always suggest flutter with 2:1 until proven otherwise. Because the flutter waves are usually biggest in leads II, III, aVF, a trick to recognizing flutter is to "mentally" erase the QRS in those leads and see what's left. In this example the saw-tooth pattern of atrial flutter becomes very apparent.

## Atrial Fibrillation



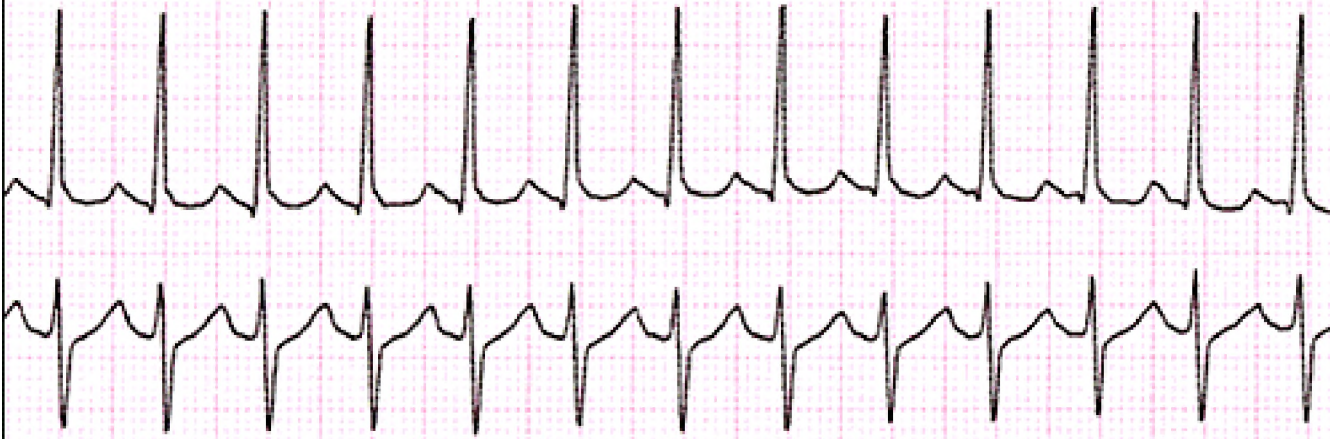
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 350-650 bpm V: Slow to rapid	Irregular	Fibrillatory (fine to course)	N/A	<.12

## Atrial Fibrillation With Moderate Ventricular Response - Marquette-KH

*Marquette Electronics Copyright 1996*



## Atrial Tachycardia

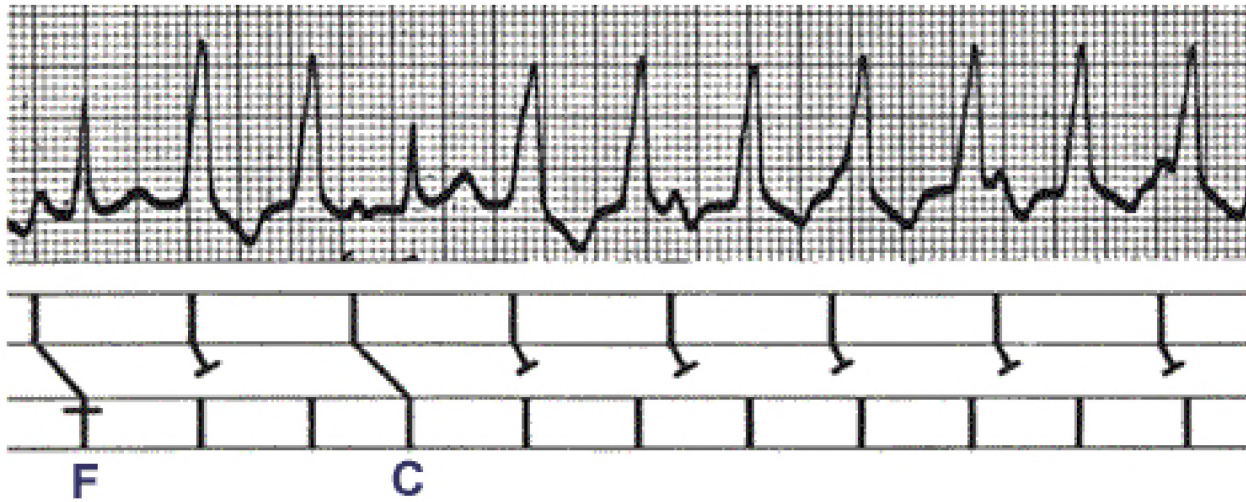


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
140-250 bpm	Regular	Abnormal P before each QRS (difficult to see)	<.20	<.12

## Atrial Tachycardia - Marquette-KH

*Marquette Electronics Copyright 1996*



**Lead II**

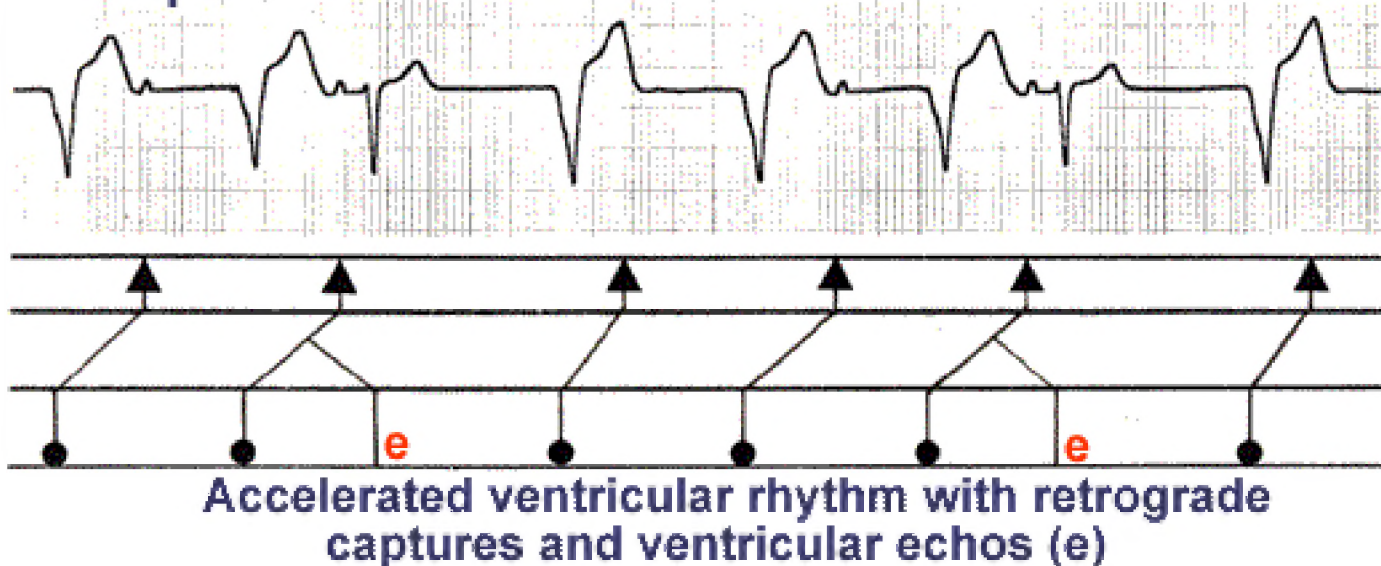
**Ventricular Tachycardia with fusions (F)  
and captures (C)**

Note: Incomplete AV dissociation

## Ventricular Tachycardia With AV Dissociation, Captures, and Fusions

*Frank Yanowitz Copyright 1996*

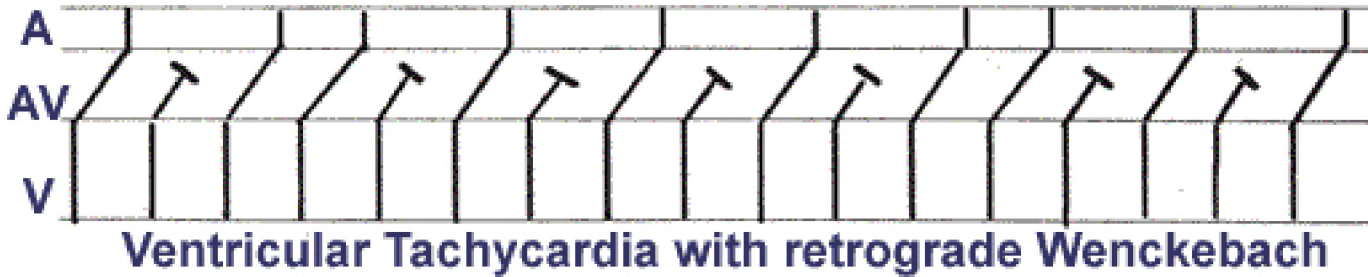
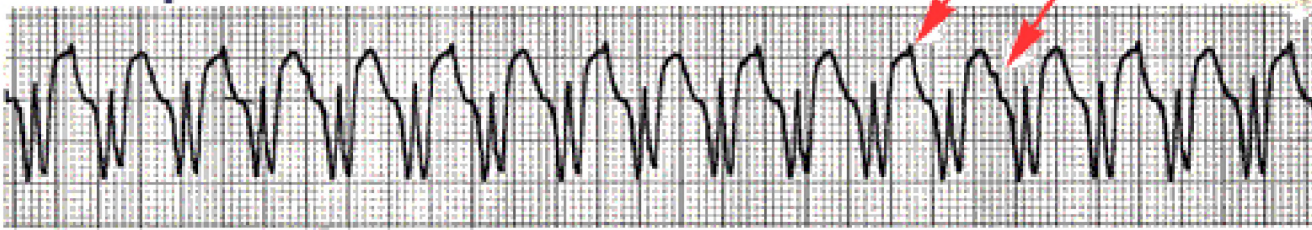
Approximately 50 percent of ventricular tachycardias are associated with AV dissociation. In these cases atrial impulses can enter the ventricles and either fuse with a ventricular ectopic beat or completely capture the ventricles. This ladder diagram illustrates these events.

Lead V<sub>1</sub>

## Accelerated Ventricular Rhythm With Retrograde Atrial Capture and Echo Beats-KH

*Frank Yanowitz Copyright 1996*

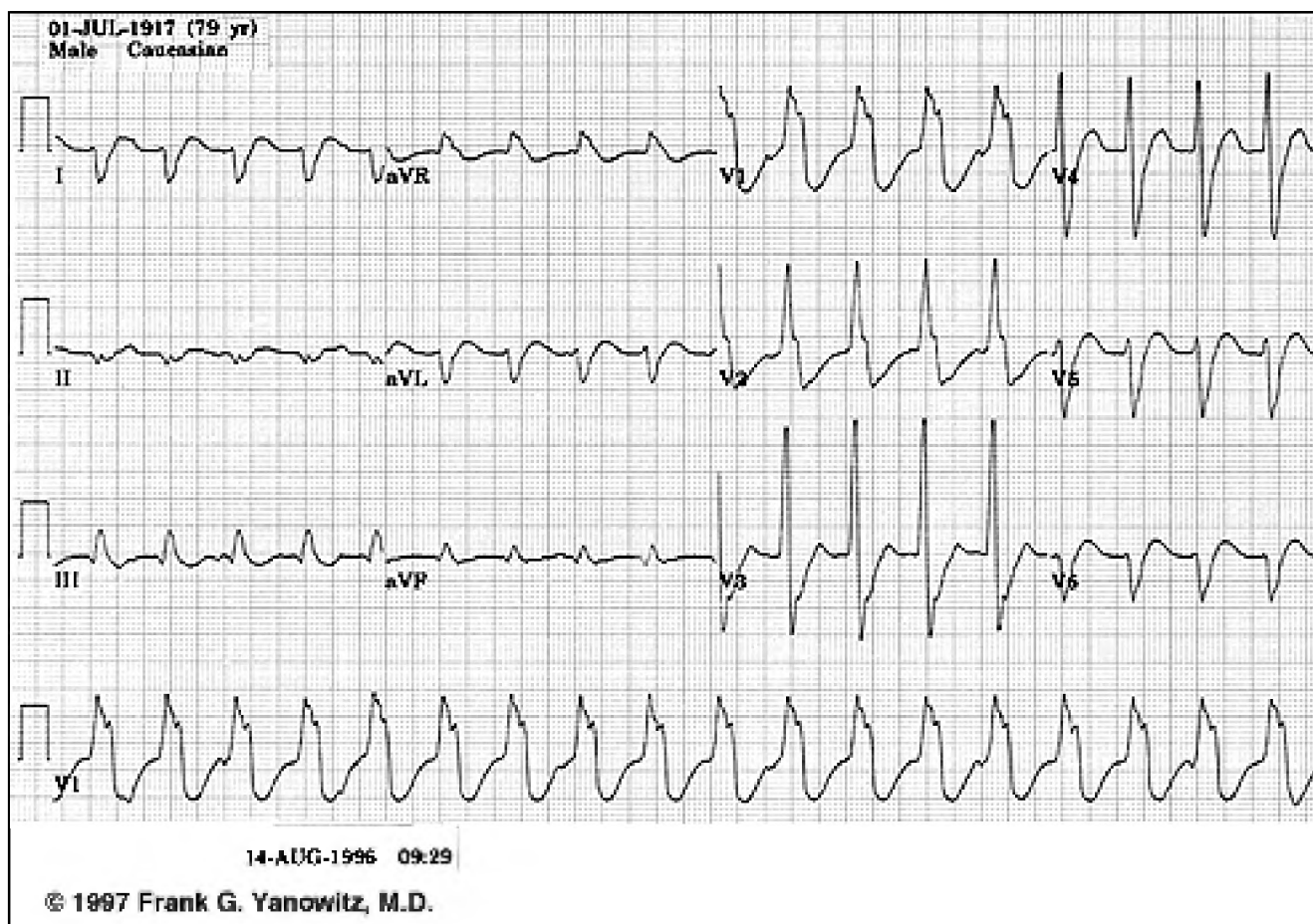
Retrograde atrial captures from an accelerated ventricular focus are occurring with increasing R-P intervals. When the longer R-P occurs, the impulse traversing the AV junction finds a route back to the ventricles, and the result is a ventricular echo.

**Lead V<sub>1</sub>**

## Ventricular Tachycardia With Retrograde Wenckebach-KH

*Frank Yanowitz Copyright 1996*

Approximately 50 percent of ventricular tachycardias are associated with AV dissociation. The other 50 percent have retrograde atrial capture. This example shows ventricular tachycardia with retrograde Wenckebach. The retrograde P waves are hard to find, but the arrows are of some help.

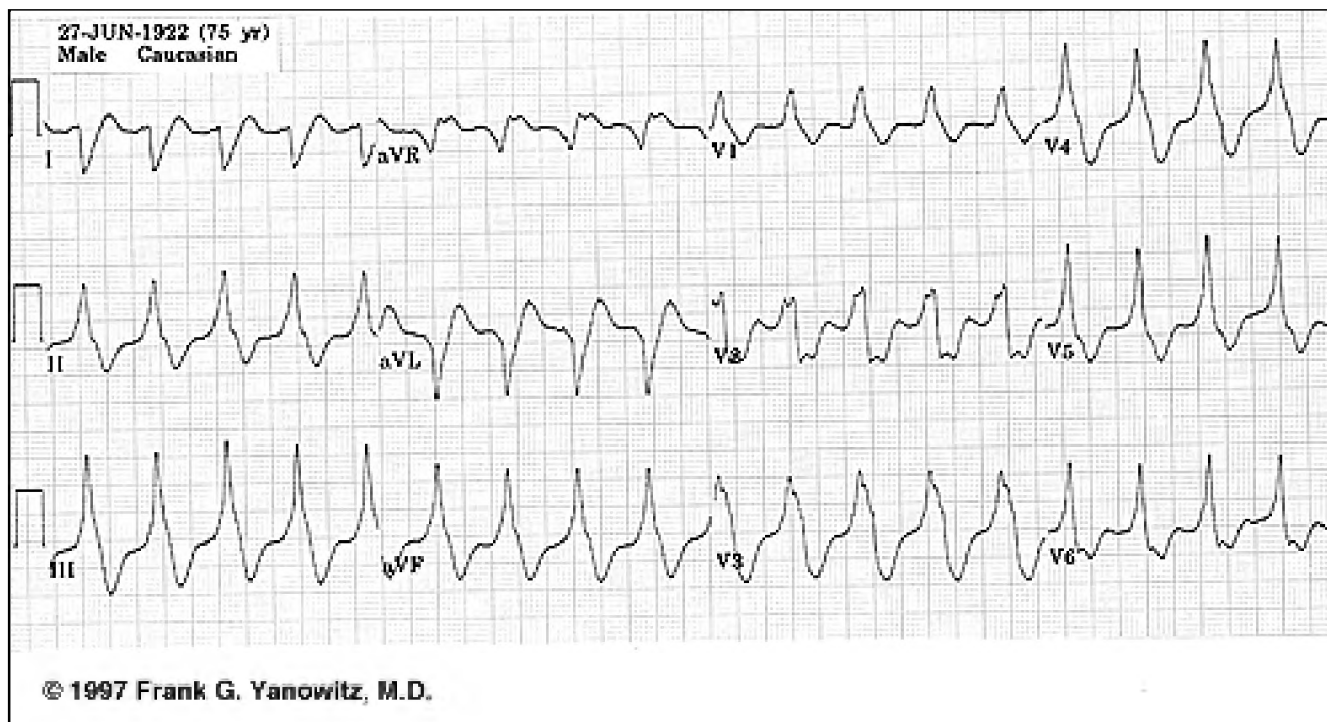


## Left Ventricular Tachycardia

*Frank G. Yanowitz, M.D. Copyright 1998*

Several features confirm this wide QRS tachycardia to be ventricular in origin. The morphology of the QRS in V1 has a distinct notch on the downstroke making it highly unlikely to be RBBB aberration. The QRS is entirely negative in lead V6. The frontal plane QRS axis is +150. The direction of ventricular activation is from left to right and posterior to anterior, suggesting a left ventricular origin.

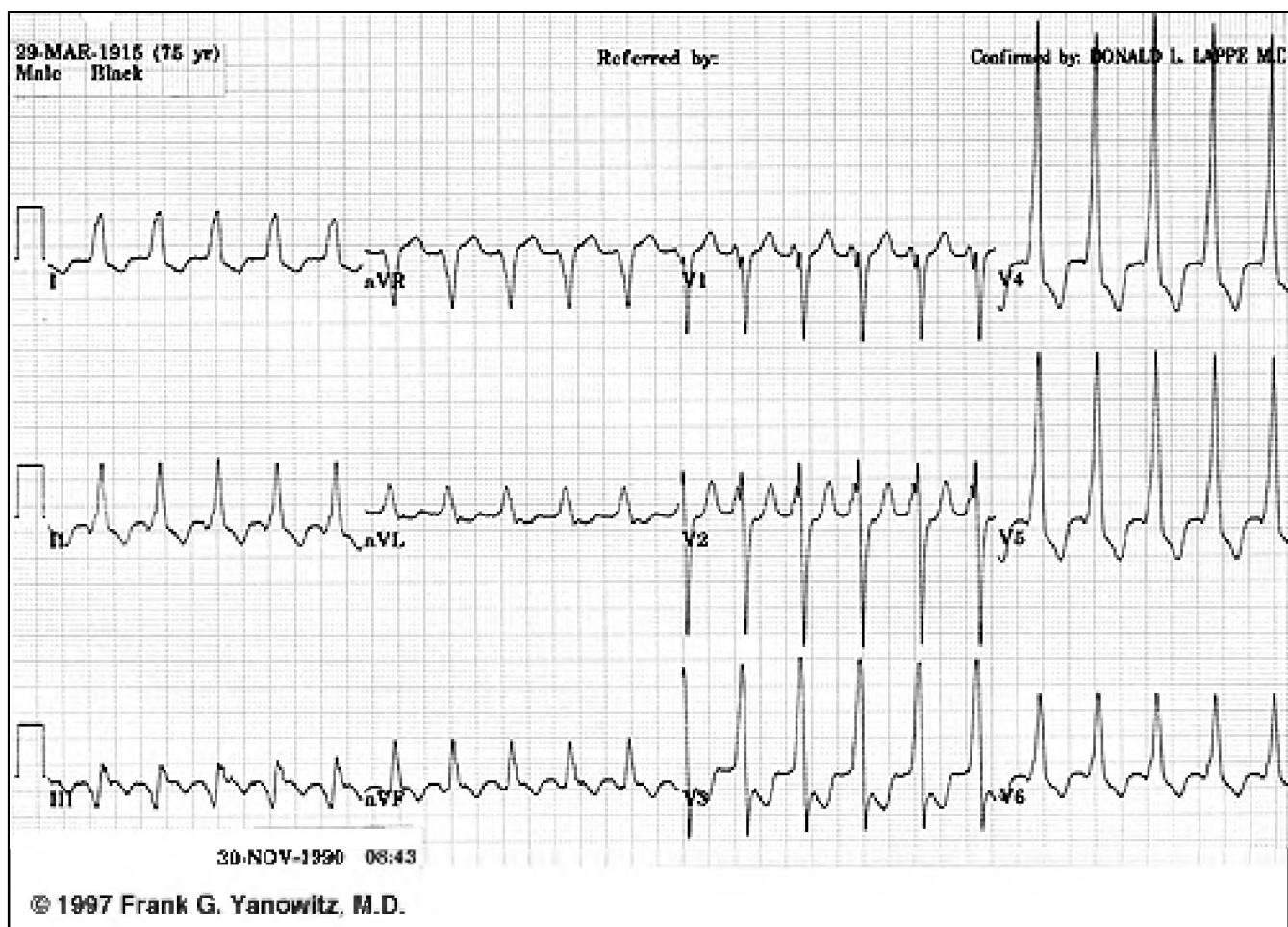




# Ventricular Tachycardia

*Frank G. Yanowitz, M.D. Copyright 1998*

The main features of this wide QRS tachycardia that indicate its ventricular origin is the concordance of QRS's in the precordial leads (all QRS's are in the same direction).



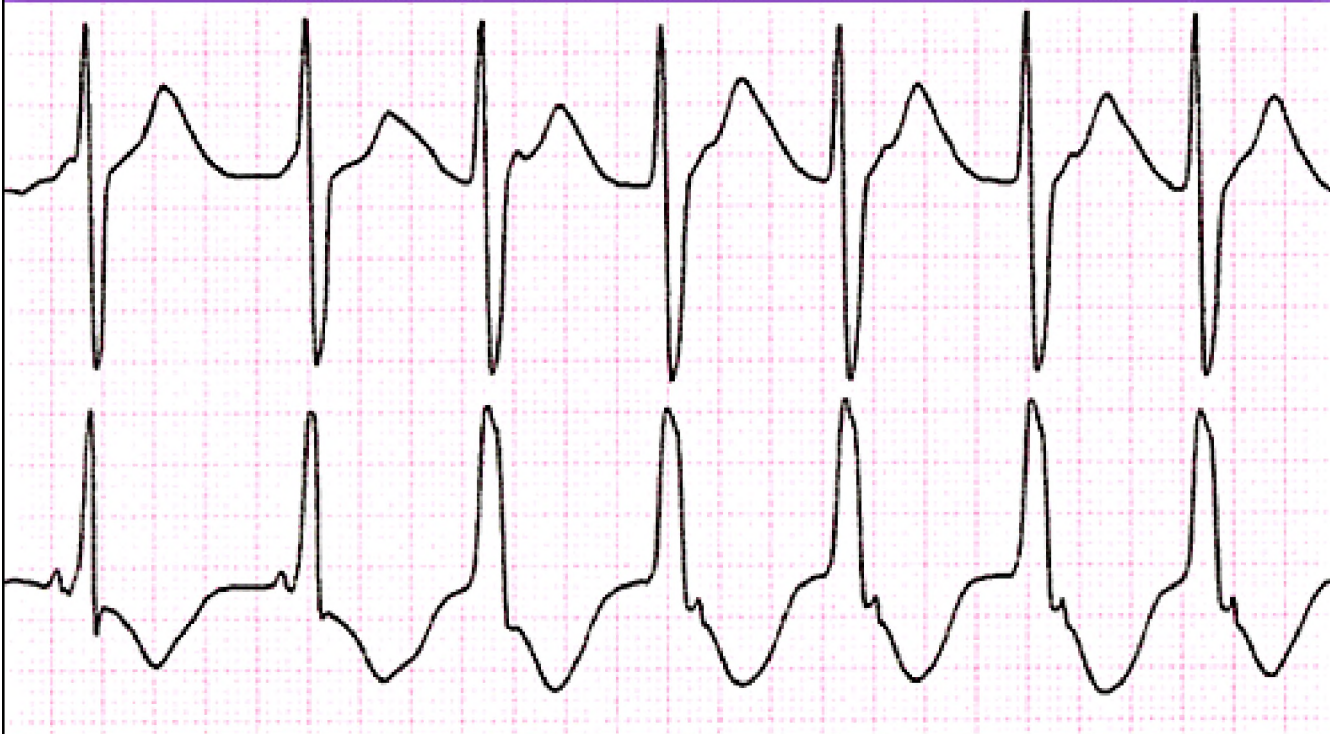
## Right Ventricular Tachycardia

*Frank G. Yanowitz, M.D. Copyright 1998*

This wide QRS tachycardia is ventricular in origin because of the classic morphology in lead V1: "fat" little r wave, notch on the downstroke of the S wave and a delay from onset of the QRS to the nadir of the S wave of  $>0.06$  s. The orientation of QRS forces is right to left and anterior to posterior, suggesting a right ventricular origin.



## Accelerated Idioventricular Rhythm (AIR)

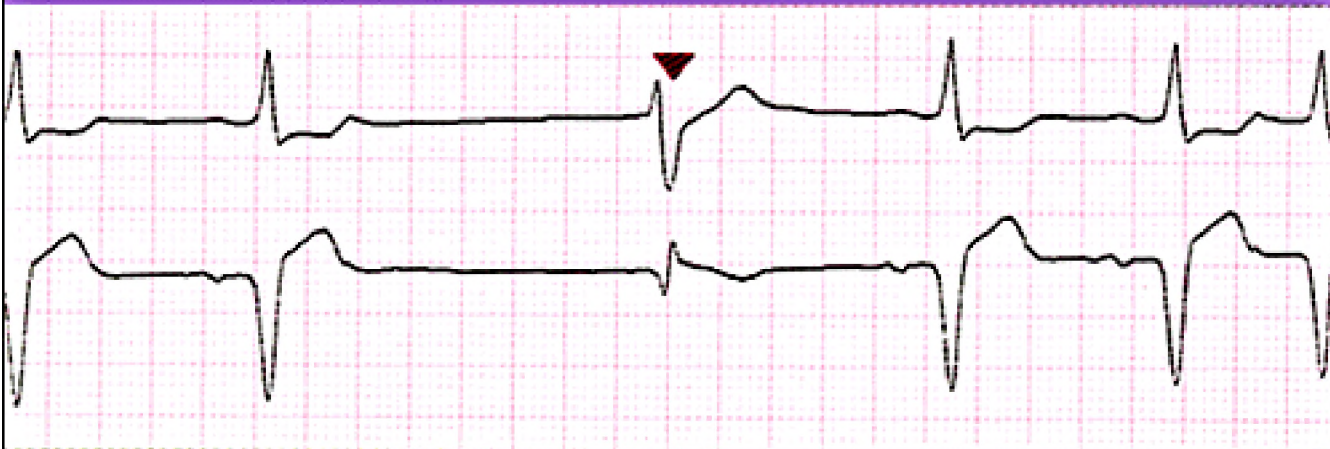


Heart Rate	Rhythm	P wave	PR interval (in seconds)	QRS (in seconds)
40-100	Regular	Absent or not related	N/A	$\geq .12$

## Accelerated IVR With AV Dissociation - Marquette-KH

*Marquette Electronics Copyright 1996*

## Ventricular Escape Beat

**Heart  
Rate****Rhythm****P Wave****PR interval  
(in seconds)****QRS  
(in seconds)**

N/A

occurs later  
than expected

Absent

N/A

 $\geq .12$ 

## Ventricular Escape Beat - Marquette-KH

*Marquette Electronics Copyright 1996*

## Idioventricular Rhythms

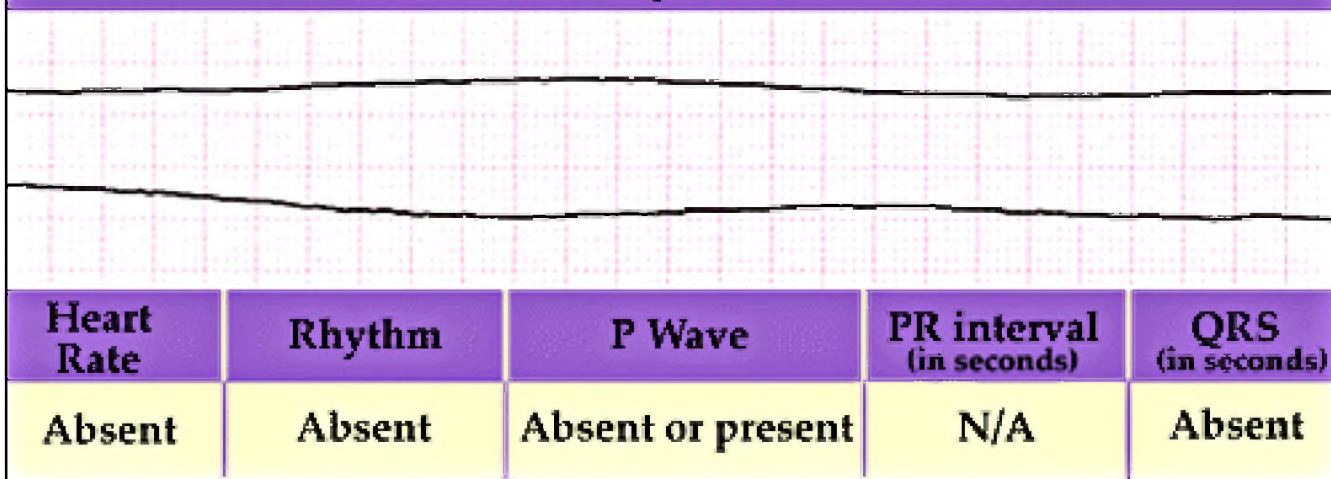


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
20-40	Regular	Absent or not related	N/A	$\geq .12$

## Idioventricular Escape Rhythm-KH

*Marquette Electronics Copyright 1996*



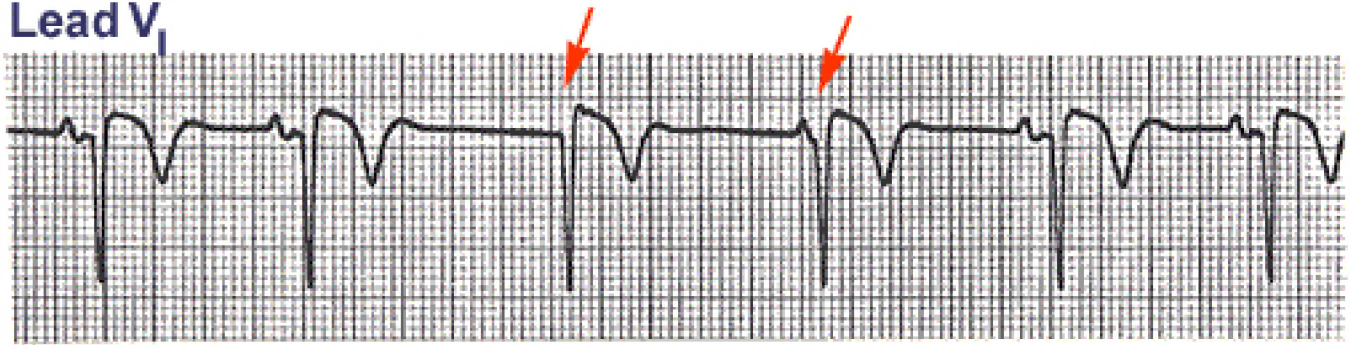
**Ventricular Asystole (standstill)**

Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
Absent	Absent	Absent or present	N/A	Absent

**Ventricular Asystole - Marquette-KH**

*Marquette Electronics Copyright 1996*

Lead V<sub>1</sub>



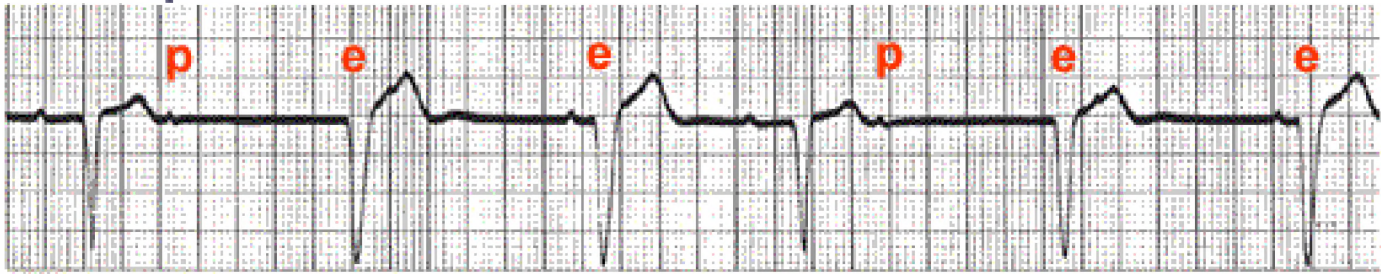
**Incomplete AV dissociation due to sinus slowing (default)  
with junctional escapes (arrows)**

## AV Dissociation by Default

*Frank Yanowitz Copyright 1996*

If the sinus node slows too much a junctional escape pacemaker may take over as indicated by arrows. AV dissociation is incomplete, since the sinus node speeds up and recaptures the entrices.

## Lead V<sub>1</sub>



**Incomplete AV dissociation (default)**

p = nonconducted PAC  
e = ventricular escapes

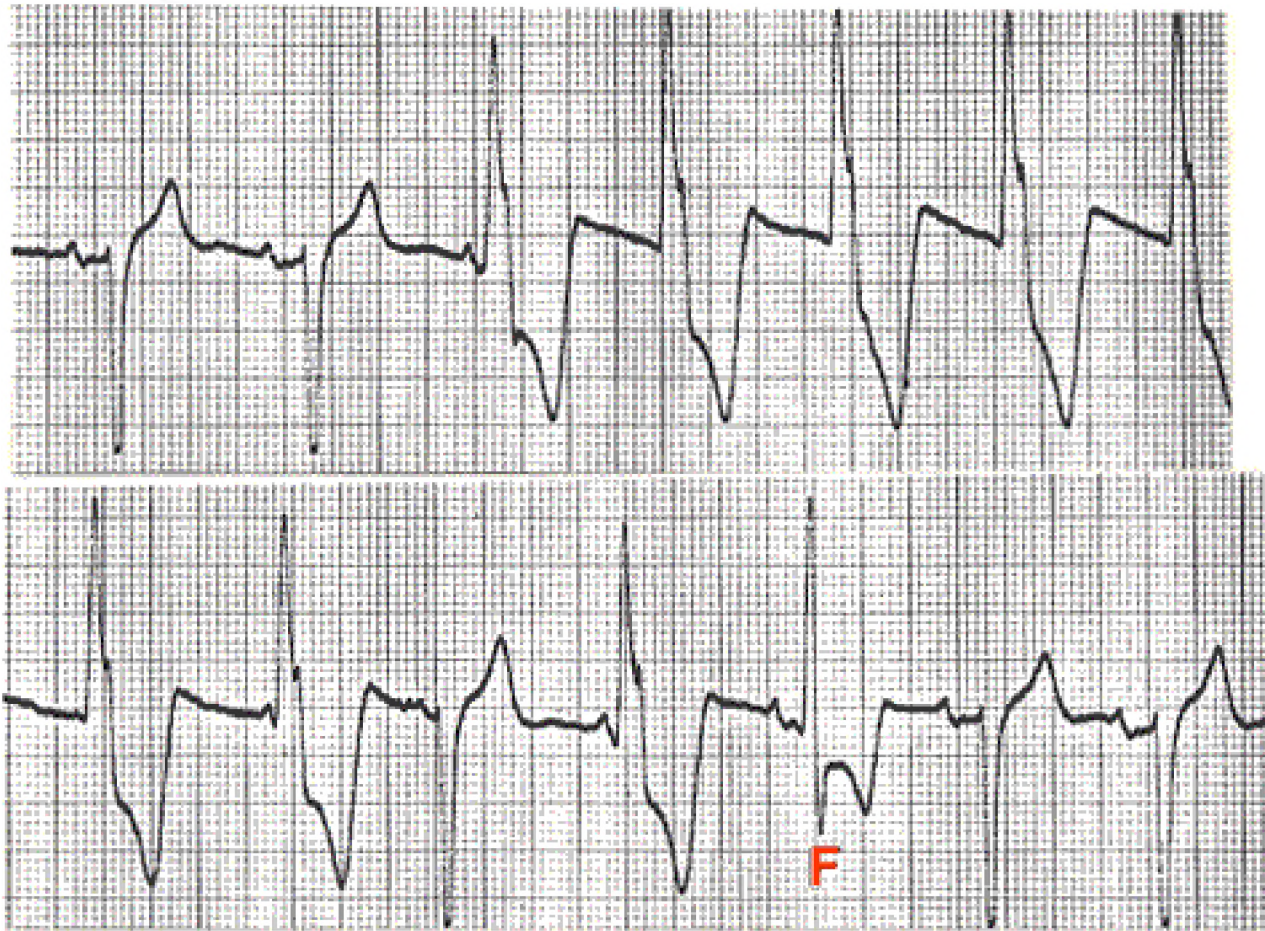
## AV Dissociation by Default

*Frank Yanowitz Copyright 1996*

The nonconducted PAC's set up a long pause which is terminated by ventricular escapes; note the wider QRS morphology of the escape beats indicating their ventricular origin. Incomplete AV dissociation occurs during the escape beats, since the atria are still under the control of the sinus node.



## Lead V<sub>1</sub>



**Incomplete AV dissociation (usurpation)  
due to accelerated ventricular rhythm**

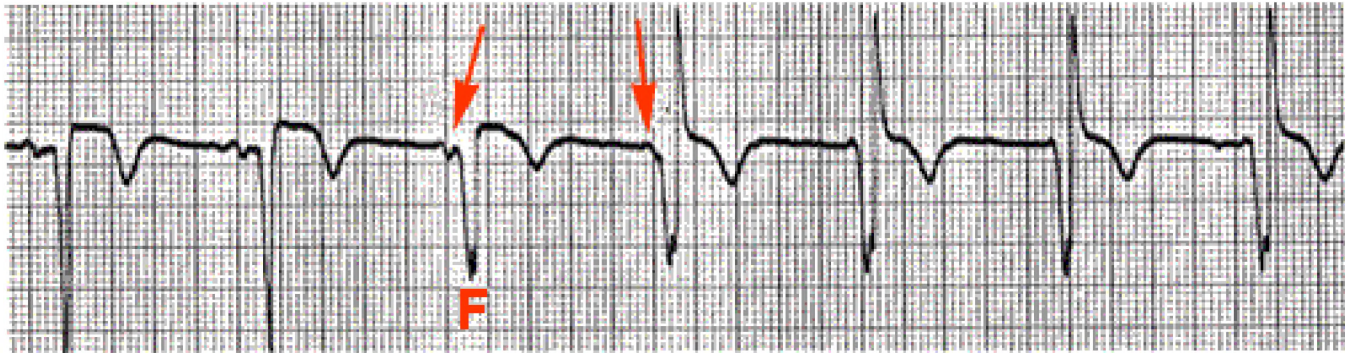
**F = fusion beat**

## AV Dissociation by Usurpation

*Frank Yanowitz Copyright 1996*

Normal sinus rhythm is interrupted by an accelerated ventricular rhythm whose rate is slightly faster than the sinus rhythm. Fusion QRS complexes occur whenever the sinus impulse enters the ventricles at the same time the ectopic ventricular focus initiates its depolarization.

## Lead MCL<sub>I</sub>



**Isochronic Ventricular rhythm**

**F = fusion beat**

**Arrows point at dissociated P waves**

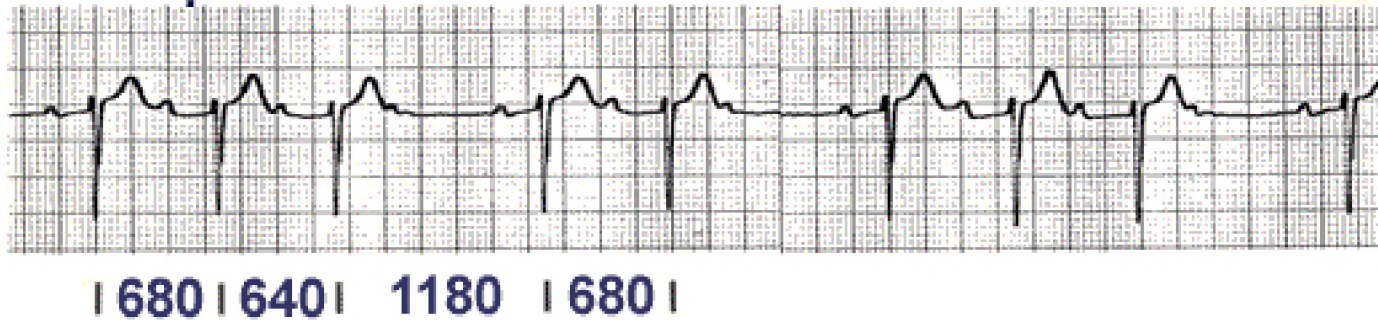
# Isochronic Ventricular Rhythm

*Frank Yanowitz Copyright 1996*

An isochronic ventricular rhythm is also called an accelerated ventricular rhythm because it represents an active ventricular focus (i.e. not an escape rhythm). This arrhythmia is a common reperfusion arrhythmia in acute MI patients. It often begins and ends with fusion beats and there is AV dissociation. Treatment is usually not necessary because the arrhythmia is self-limiting.



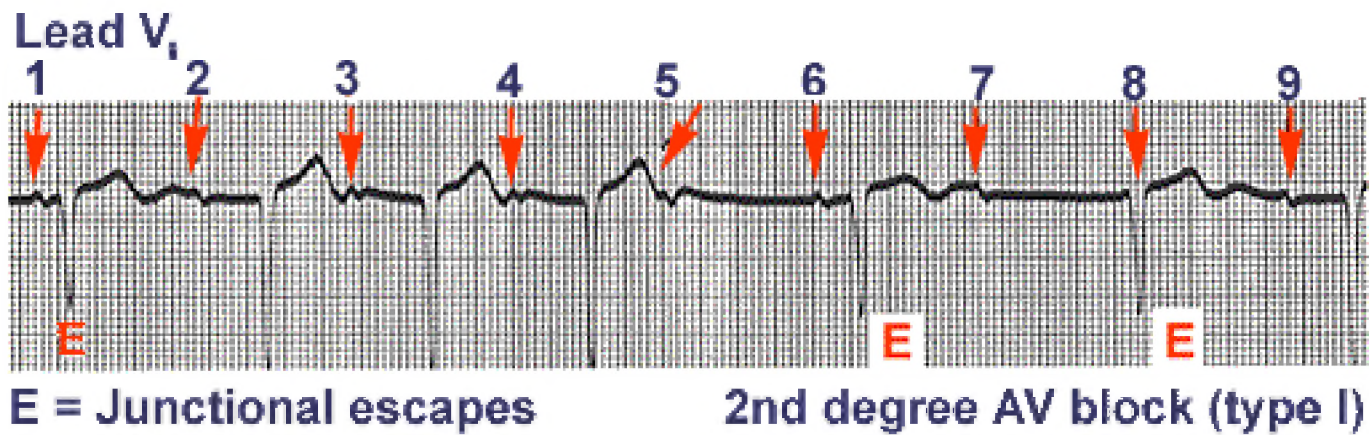
## Lead V<sub>1</sub> "Classic Wenckebach"



## 2nd Degree AV Block, Type I

*Frank Yanowitz Copyright 1996*

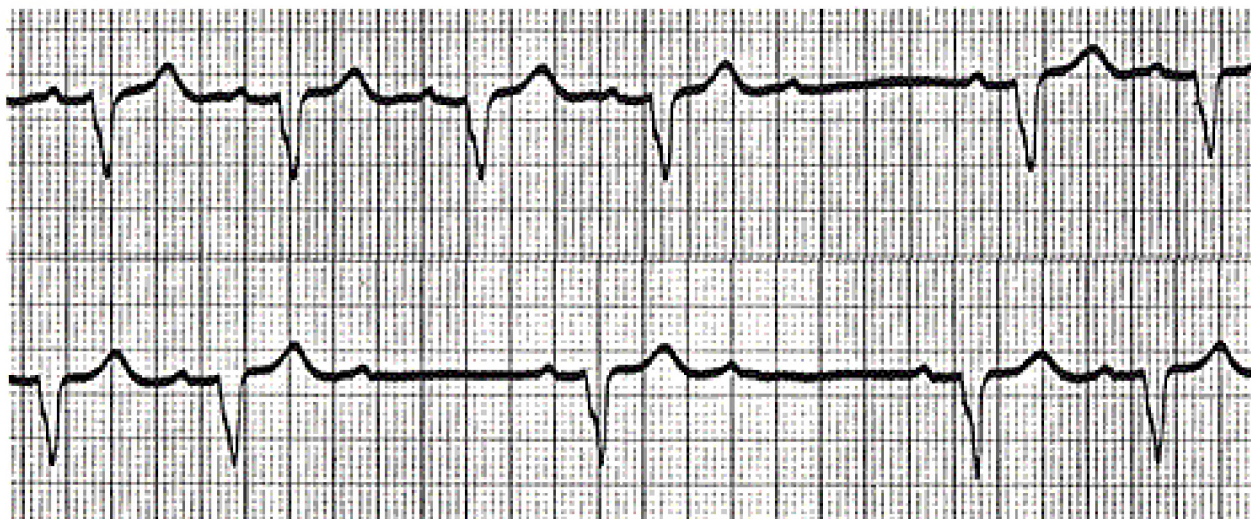
The 3 rules of "classic AV Wenckebach" are: 1. decreasing RR intervals until pause; 2. the pause is less than preceding 2 RR intervals; and 3. the RR interval after the pause is greater than the RR interval just prior to pause. Unfortunately, there are many examples of atypical forms of Wenckebach where these rules don't hold.



## 2nd Degree AV Block, Type I, with Junctional Escapes

*Frank Yanowitz Copyright 1996*

Junctional escapes are passive, protective events whenever the heart rate slows below that of the escape mechanism. In this example of 2nd degree AV block, type I, the escapes occur following the non-conducted P waves. Arrows indicate the position of the P waves. Note that the escape beats have a slightly different QRS morphology than the conducted sinus beats.

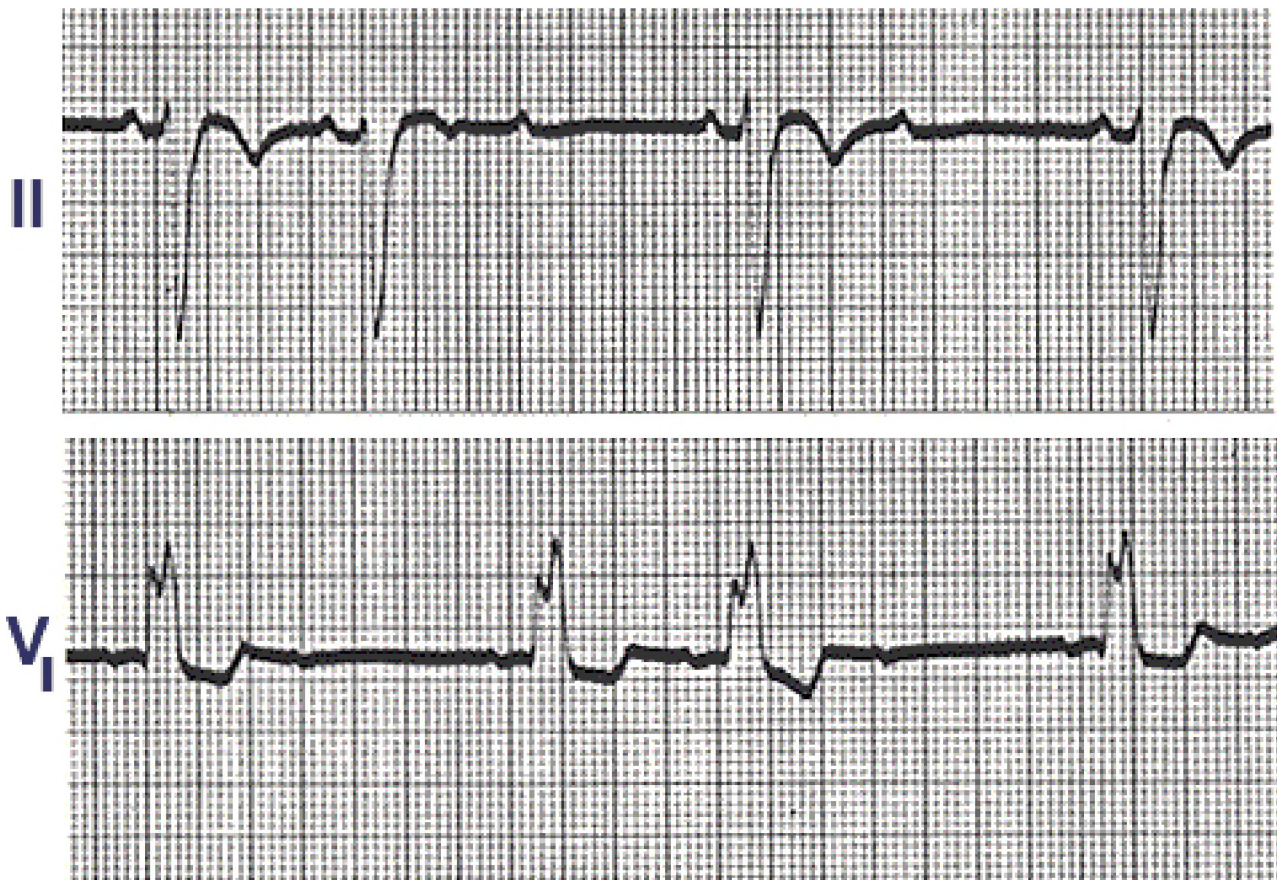
**Lead V<sub>1</sub>****Diagnosis?**

## **LBBB and 2nd degree AV Block, Mobitz Type II**

*Frank Yanowitz Copyright 1996*

Mobitz II 2nd degree AV block is usually a sign of bilateral bundle branch disease. One of the two bundle branches should be completely blocked; in this example the left bundle is blocked. The nonconducted sinus P waves are most likely blocked in the right bundle which exhibits 2nd degree block. Although unlikely, it is possible that the P waves are blocked somewhere in the AV junction such as the His bundle.





Type II AV block. How many fascicles are in trouble?

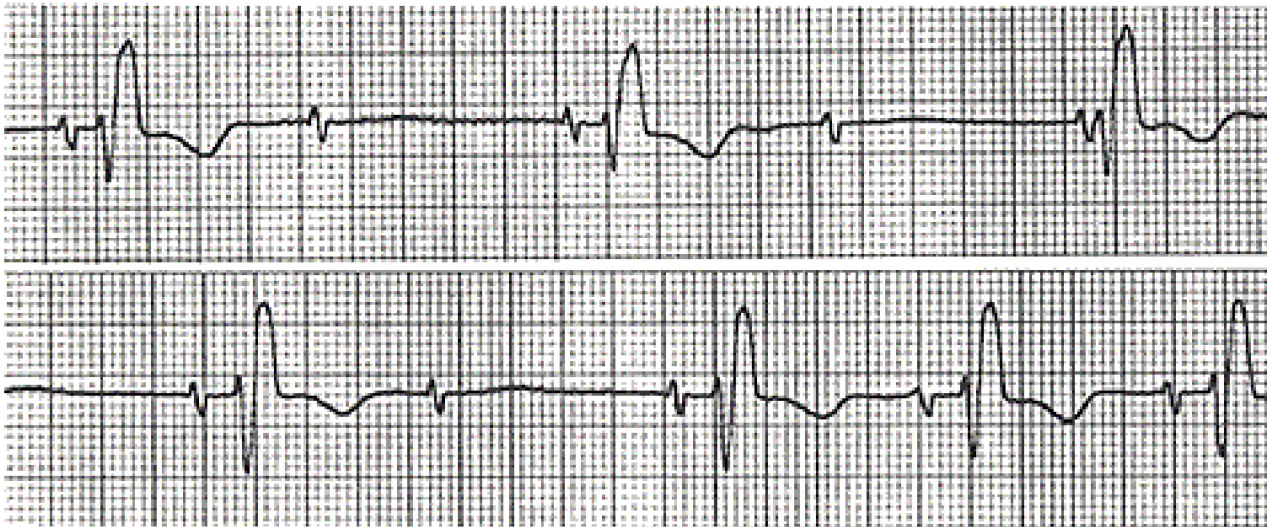
## Trifascicular Block: RBBB, LAFB, and Mobitz II 2nd Degree AV Block

*Frank Yanowitz Copyright 1996*

A nice example of trifascicular block: Lead V1 shows RBBB; Lead II is mostly negative with an rS morphology suggesting left anterior fascicular block. Since Mobitz II 2nd degree AV block is more often located in the bundle branch system, the only location left for this block is the left posterior division of the left bundle. Therefore all three ventricular conduction pathways are diseased.



## Lead V<sub>1</sub>



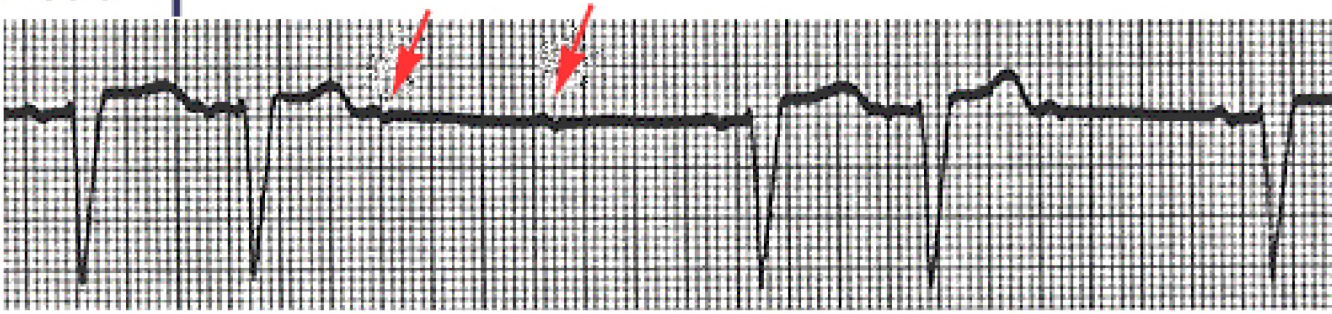
**Where is the block?**

## RBBB plus Mobitz II 2nd Degree AV Block

*Frank Yanowitz Copyright 1996*

The classic rSR' in V<sub>1</sub> is RBBB. Mobitz II 2nd degree AV block is present because the PR intervals are constant. Statistically speaking, the location of the 2nd degree AV block is in the left bundle branch rather than in the AV junction. The last QRS in the top strip is a junctional escape, since the PR interval is too short to be a conducted beat.

## Lead V<sub>1</sub>

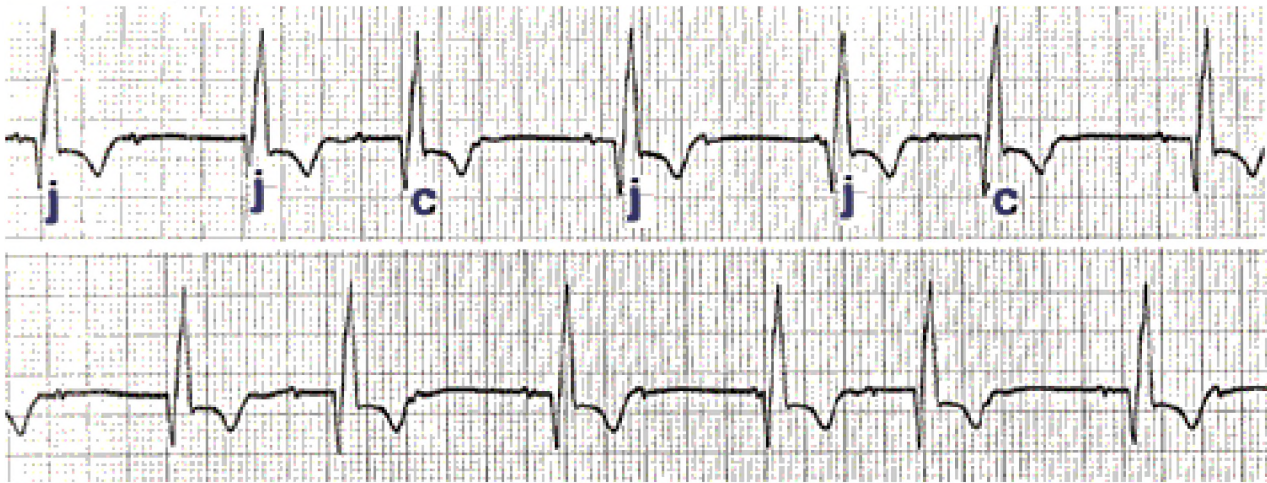


## Mobitz II 2nd Degree AV Block With LBBB

*Frank Yanowitz Copyright 1996*

The QRS morphology in lead V<sub>1</sub> shows LBBB. The arrows point to two consecutive nonconducted P waves, most likely hung up in the diseased right bundle branch. This is classic Mobitz II 2nd degree AV block.

## Lead V<sub>1</sub>

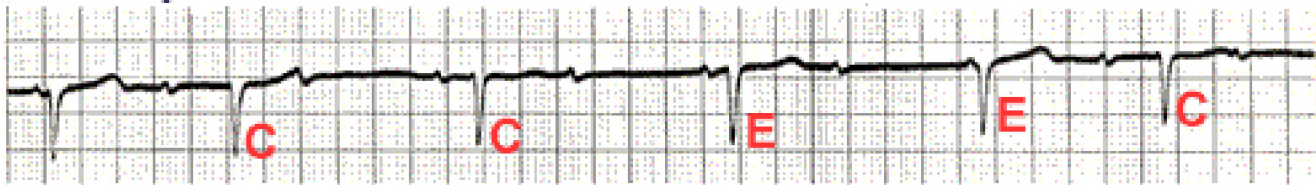


**Diagnosis?**

# Incomplete AV Dissociation Due To 2nd Degree AV Block

*Frank Yanowitz Copyright 1996*

2nd degree AV block is evident from the nonconducted P waves. Junctional escapes, labeled 'J', terminate the long pauses because that's the purpose of escape pacemakers....to protect us from too slow heart rates. All QRS's with shorter RR intervals are capture beats, labeled 'c'. Atypical RBBB with a qR pattern suggests a septal MI.

**Lead V<sub>1</sub>****High grade 2nd degree AV block (type I)****C = Captures****E = Junctional escapes**

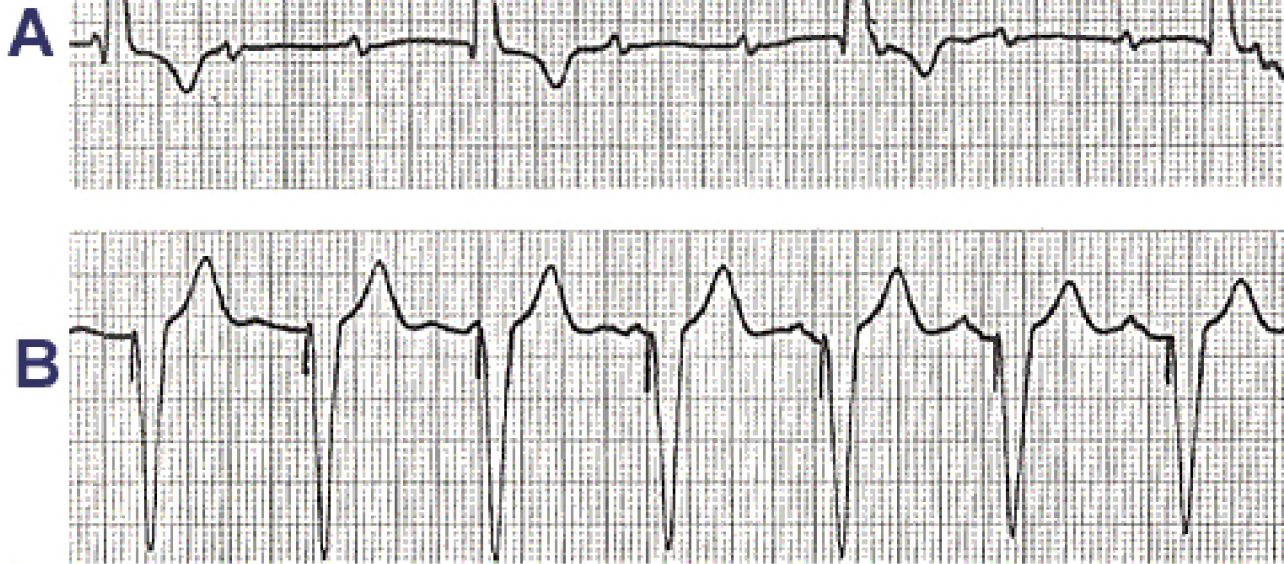
## 2nd Degree AV Block, Type I With Escapes and Captures

*Frank Yanowitz Copyright 1996*

Often in the setting of 2nd degree AV block the pauses caused by nonconducted P waves are long enough to enable escape pacemakers from the junction or ventricles to take over. This example illustrates junctional escapes, labeled 'E' and captures, labeled 'C'. Note that the PR intervals for the captures vary, making this Type I 2nd degree AV block. AV dissociation is seen when the escape beats occur.



## Lead V<sub>1</sub>

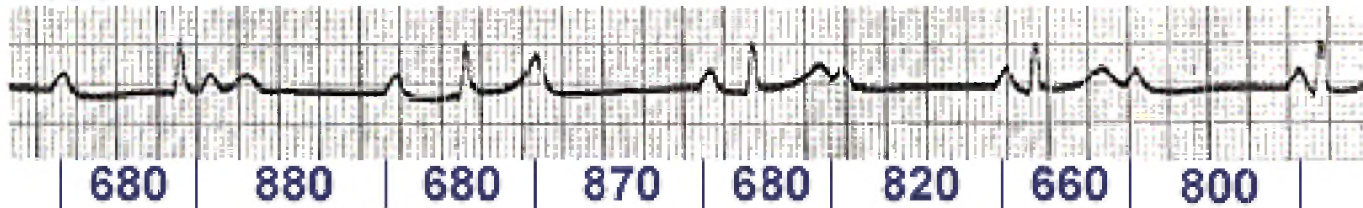


## 3rd Degree AV Block Rx'ed With a Ventricular Pacemaker

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In 'A' the ECG shows complete or 3rd degree AV block with a left ventricular escape rhythm, as evidenced by the upright QRS morphology. In 'B' the artificial right ventricular pacemaker rhythm is shown.



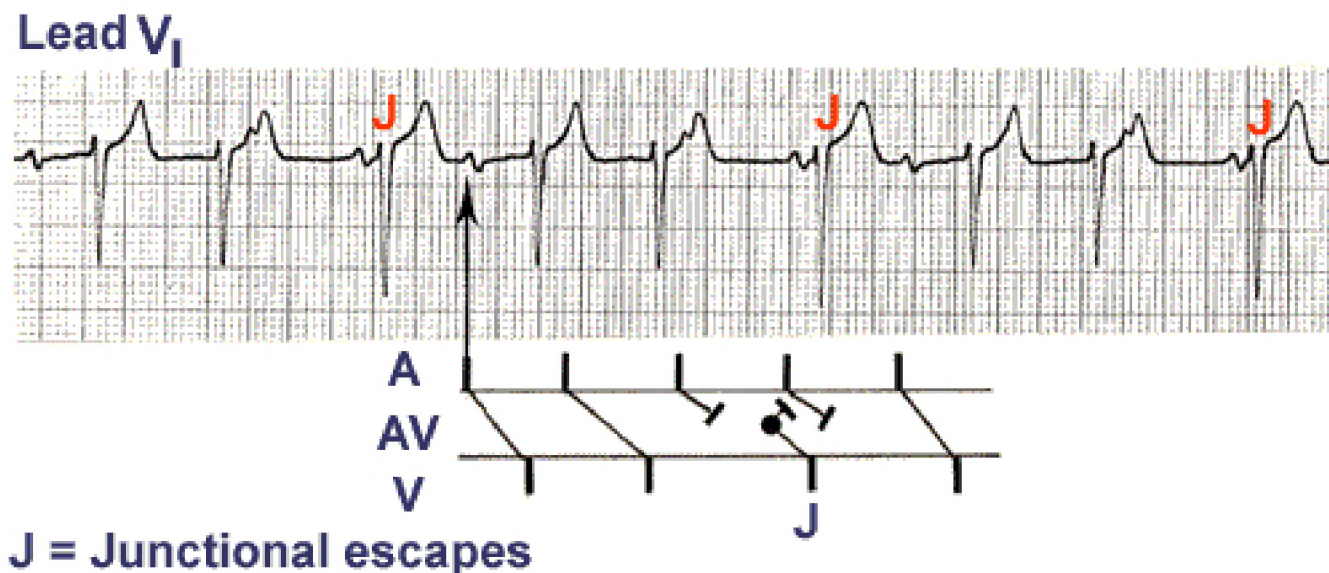
**Lead II**

1. What is the diagnosis?
2. Why are the PP intervals alternating?

## Complete AV Block, Junctional Escape Rhythm, and Ventriculophasic Sinus Arrhythmia

*Frank Yanowitz Copyright 1996*

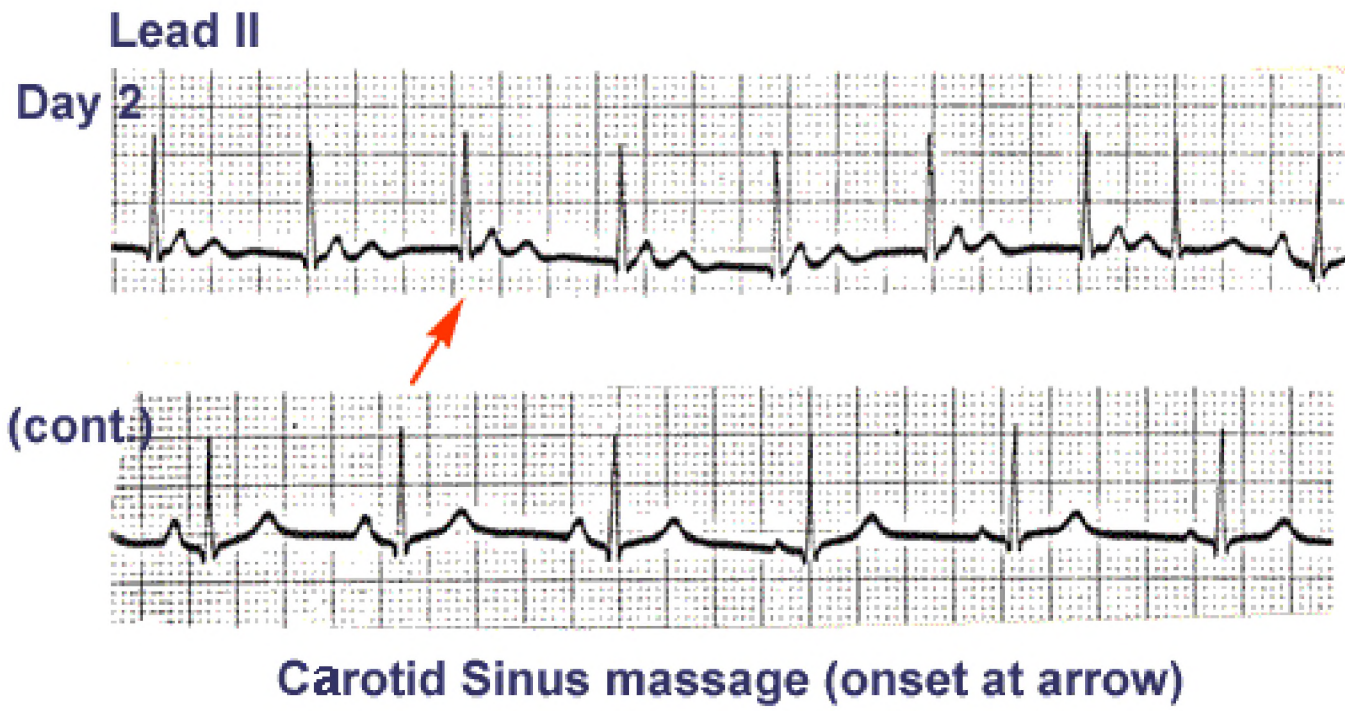
Complete AV block is seen as evidenced by the AV dissociation. A junctional escape rhythm sets the ventricular rate at 45 bpm. The PP intervals vary because of ventriculophasic sinus arrhythmia; this is defined when the PP interval that includes a QRS is shorter than a PP interval that excludes a QRS. The QRS generates a strong enough pulse to activate the carotid sinus mechanism which slows the subsequent PP interval.



## 2nd Degree AV Block, Type I, With Accelerated Junctional Escapes and a Ladder Diagram

*Frank Yanowitz Copyright 1996*

The ladder diagram illustrates a Wenckebach type AV block by the increasing PR intervals before the blocked P wave. After the blocked P wave, however, a rev-ed up junctional pacemaker terminates the pause. Note that the junctional beats have a slightly different QRS morphology from the sinus beats making them more easily recognized. Note also the AV dissociation that accompanies the junctional beats.

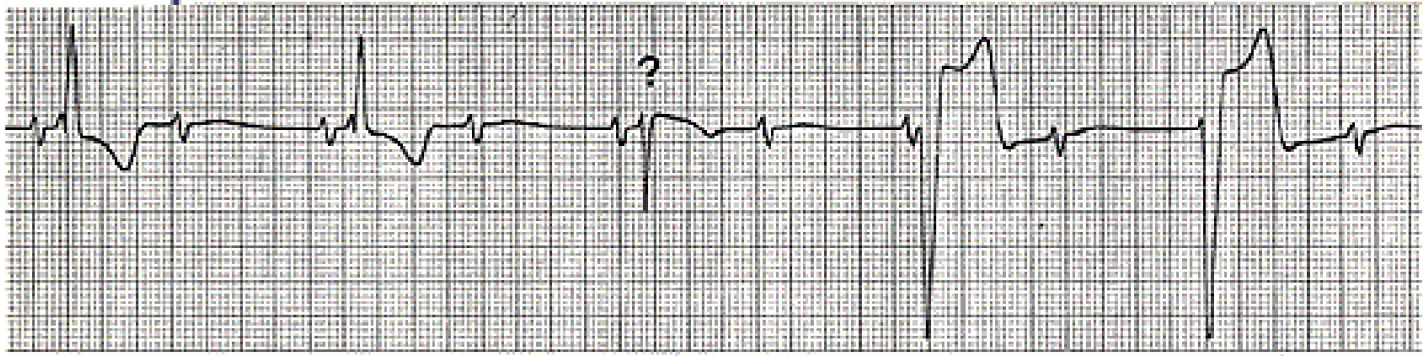


## ECG Of The Century - Part II: Dual AV Pathways

*Frank Yanowitz Copyright 1996*

An astute cardiology fellow, yours truly, went to the patient's bedside on Day 2 and massaged the right carotid sinus as indicated by the arrow. Four beats later at a slightly slower heart rate the PR interval suddenly normalized suggesting an abrupt change from a slow AV nodal pathway to a fast AV nodal pathway, demonstrating the existence of dual AV pathways.

## Lead V<sub>1</sub>



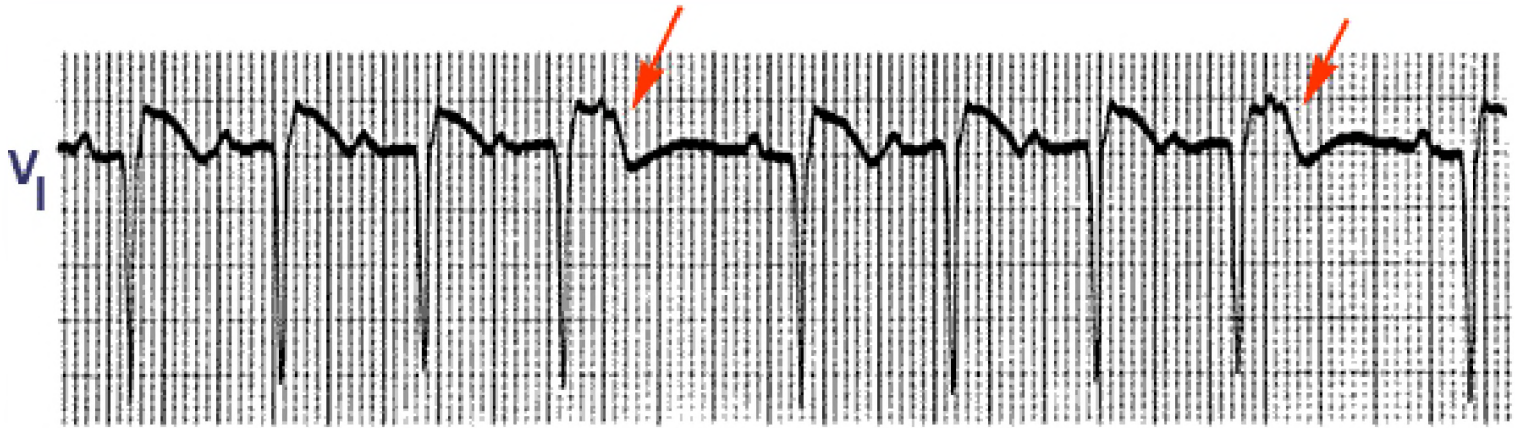
**A question (?) of morality**

## Two Wrongs Sometimes Make A Right

*Frank Yanowitz Copyright 1996*

The question mark is over a "normal" looking QRS that occurs during 2:1 AV block with RBBB. Following this QRS a ventricular escape rhythm takes over. The "normal" looking beat is actually a fusion beat resulting from simultaneous activation of the ventricles; the sinus impulse enters the left ventricle at the same time a right ventricular escape rhythm begins.





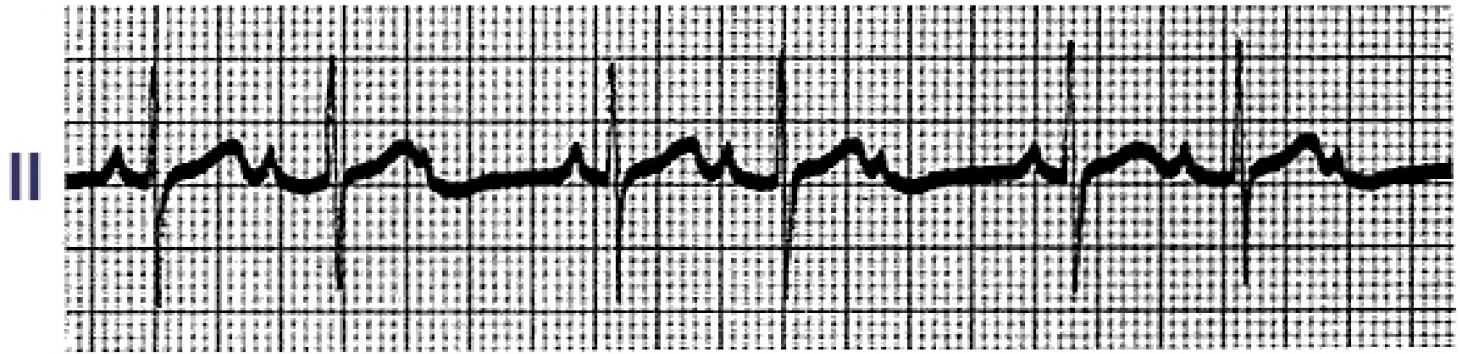
**PAC's which interrupt Wenckebach sequences and always follow long PR intervals -- atrial echos**

## Atrial Echos-KH

*Frank Yanowitz Copyright 1996*

In this example a typical Wenckebach sequence is interrupted by what looks like a PAC - indicated by red arrows. Atrial echos are more likely, however, because the preceding beat has a long PR interval, a condition that facilitates reentry and echo formation.



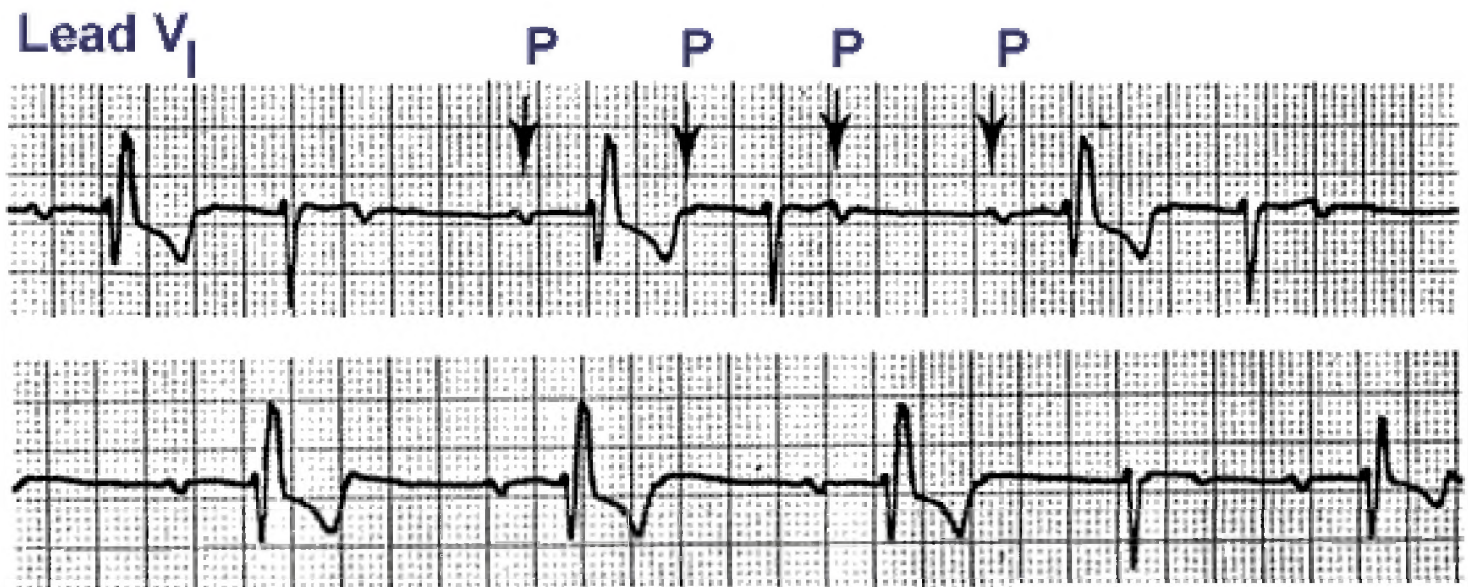


**3:2 2nd Degree AV block (Type I)**

## **Second Degree AV Block, Type I, With 3:2 Conduction Ratio-KH**

*Frank Yanowitz Copyright 1996*

There are two types of 2nd degree AV Block. In this example of Type I or Wenckebach AV block there are 3 P waves for every 2 QRS's; the PR interval increases until a P wave fails to conduct. This is an example of "group beating".



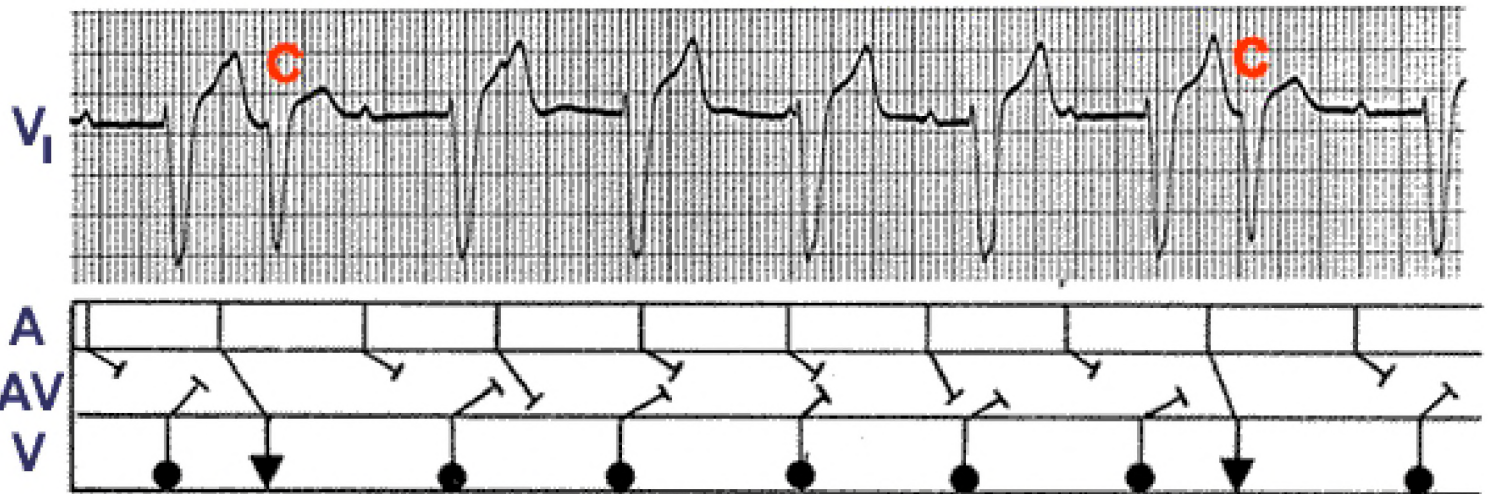
**Name 2 levels of heart block**

## **Second Degree AV Block, Type I, With Bradycardia-dependent RBBB -KH**

*Frank Yanowitz Copyright 1996*

An interesting and unusual form of rate-dependent bundle branch block. Normal sinus rhythm at 85 bpm is present with a 3:2 and 2:1 2nd degree AV block. The progressive PR prolongation in the 3:2 block makes this a type-I or Wenckebach block. Long cycles end in RBBB; short cycles have normal QRS duration. This is, therefore, a Bradycardia-dependent RBBB. The mechanism is thought to be due to latent pacemaker activity in the right bundle partially depolarizing the bundle, thus making conduction down it more difficult.

**"Supernormal Conduction":** Conduction that is better than expected under the circumstances



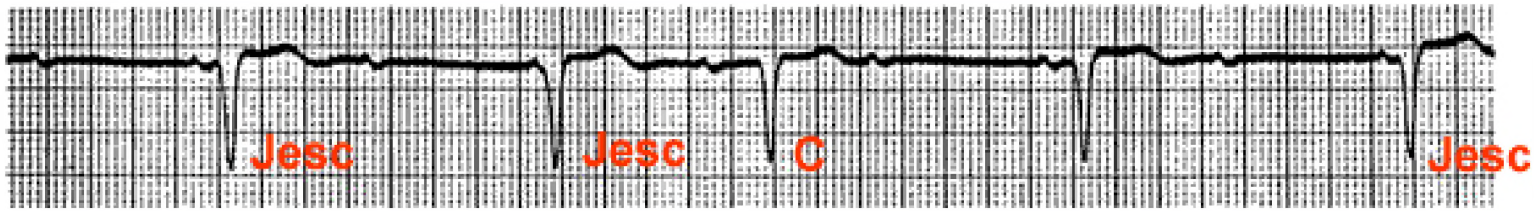
**Dx: Incomplete AV dissociation; accelerated ventricular rhythm, 2nd degree AV block with rare captures (C)**

## Supernormal Conduction: 2nd Degree AV Block With Rare Captures; Accelerated Ventricular Rhythm-KH

*Frank Yanowitz Copyright 1996*

This complicated rhythm strip illustrates "supernormal" conduction... a situation where conduction is better than expected. The ladder diagram shows that the accelerated ventricular rhythm prevents most of the sinus impulses from reaching the ventricles. Only appropriately timed sinus impulses reach the ventricle - indicated by the 'C' or capture beats. Supernormal conduction doesn't mean "better than normal", just the appearance of conducted beats when not expected.

## Lead V<sub>1</sub>



**2nd degree AV block with junctional escapes and captures**

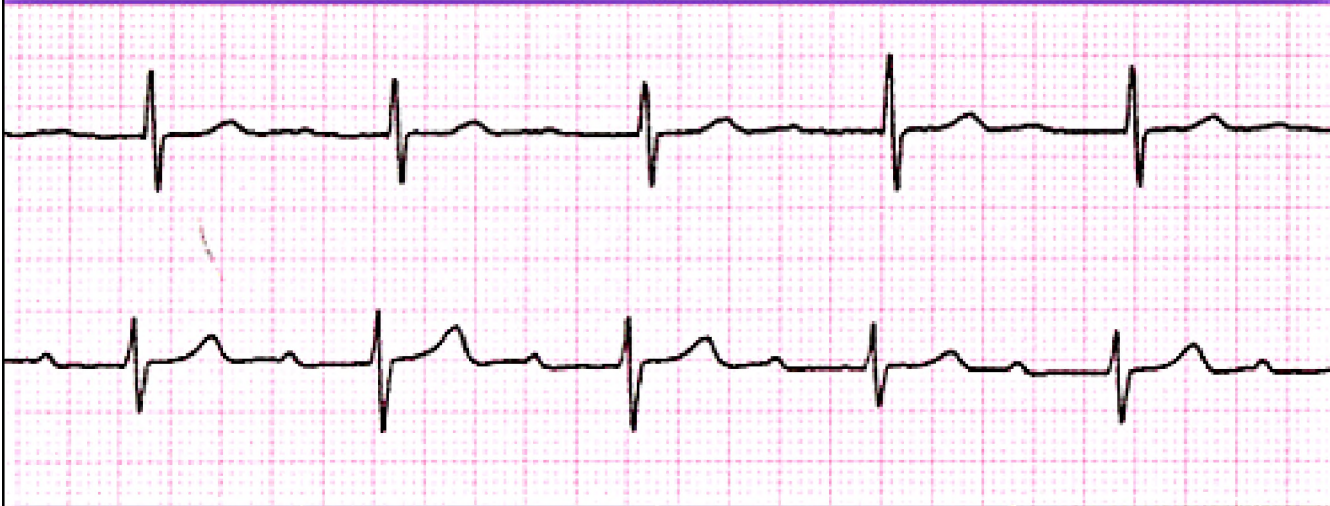
## 2nd Degree AV Block With Junctional Escapes And Captures-KH

*Frank Yanowitz Copyright 1996*

Second degree AV block is present; conducted beats are identified by those QRS's that terminate shorter cycles than the junctional escape cycle; i.e., the 3rd and probably the 4th QRS's are captures; the other QRS's are junctional escapes.



## First Degree AV Block



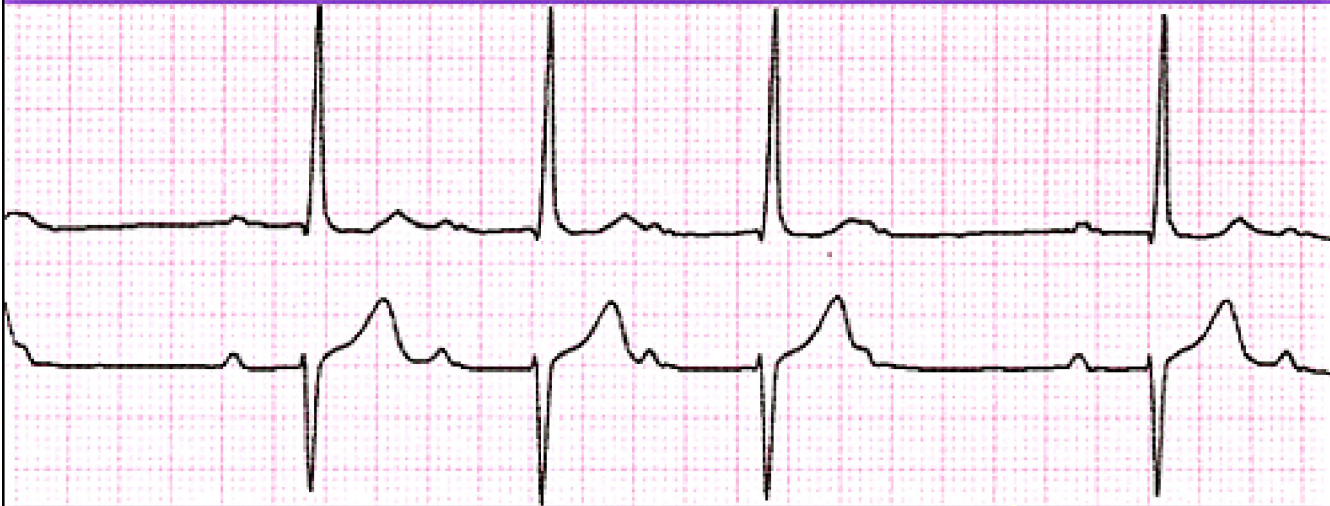
P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	>.20	>.12	Regular rhythm

## First Degree AV Block - Marquette-KH

*Marquette Electronics Copyright 1996*



## Second Degree AV Block • Mobitz 1 (Wenckebach)

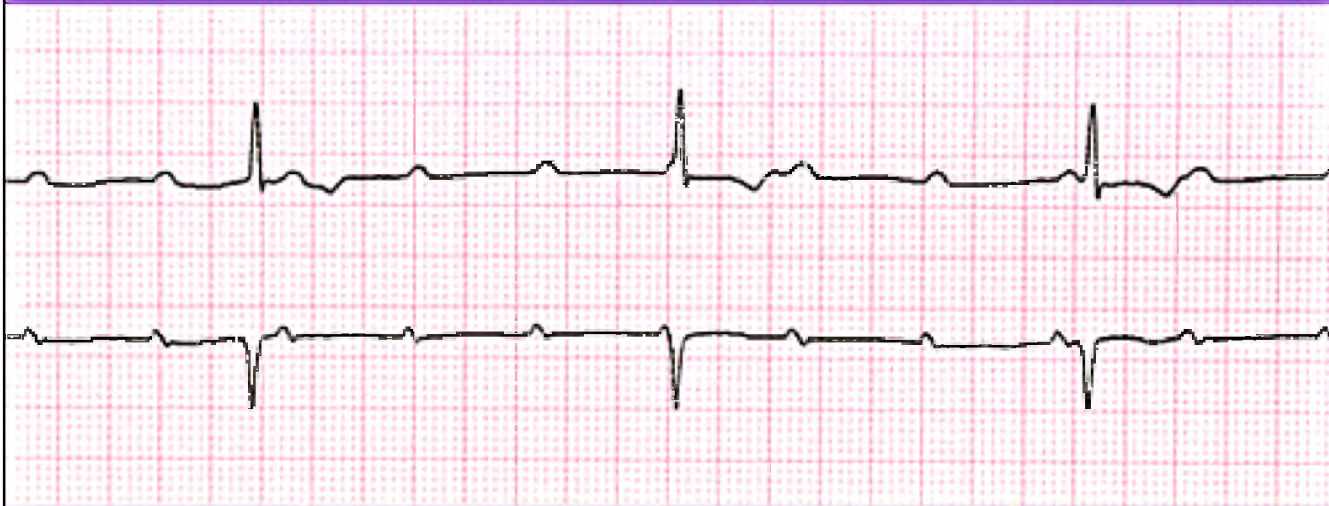


P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Conduction intermittant	Increasingly Prolonged	<.12	QRS dropped in a repeating pattern

## 2nd Degree AV Block, Type I (Wenckebach)-KH

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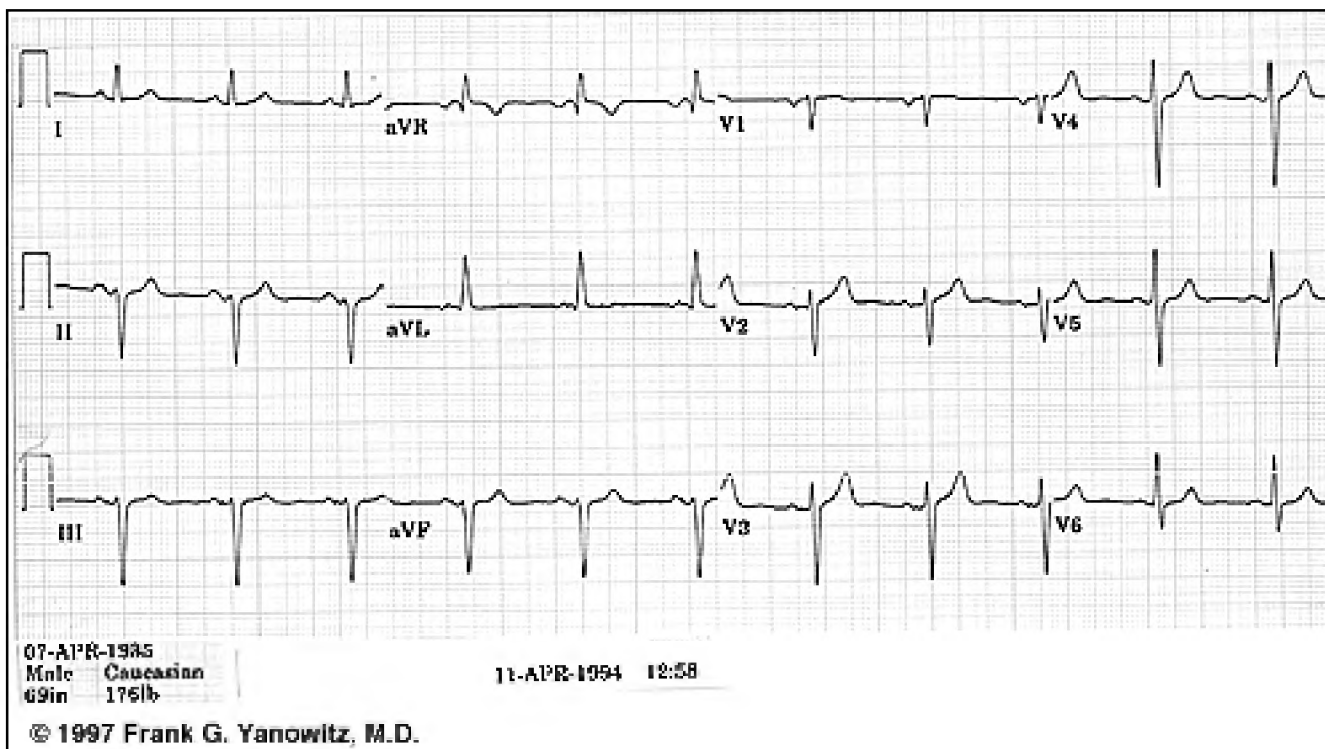
## Third Degree (complete) AV Block



P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Normal but not related to QRS	None	N/A	No relationship between P&RS

## Complete AV Block (3rd Degree) with Junctional Rhythm-KH

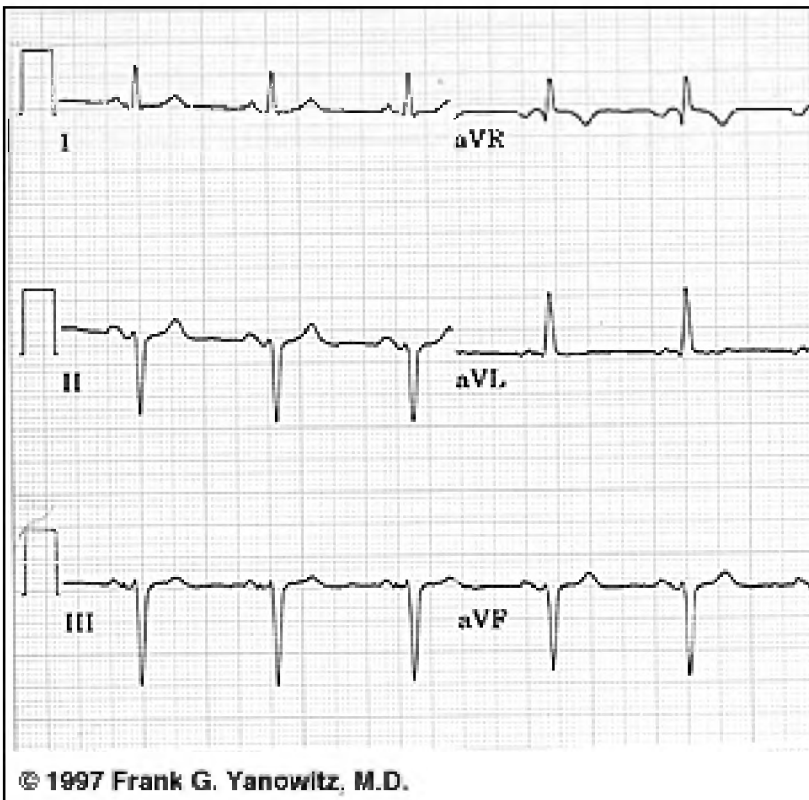
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## Left Anterior Fascicular Block (LAFB)-KH

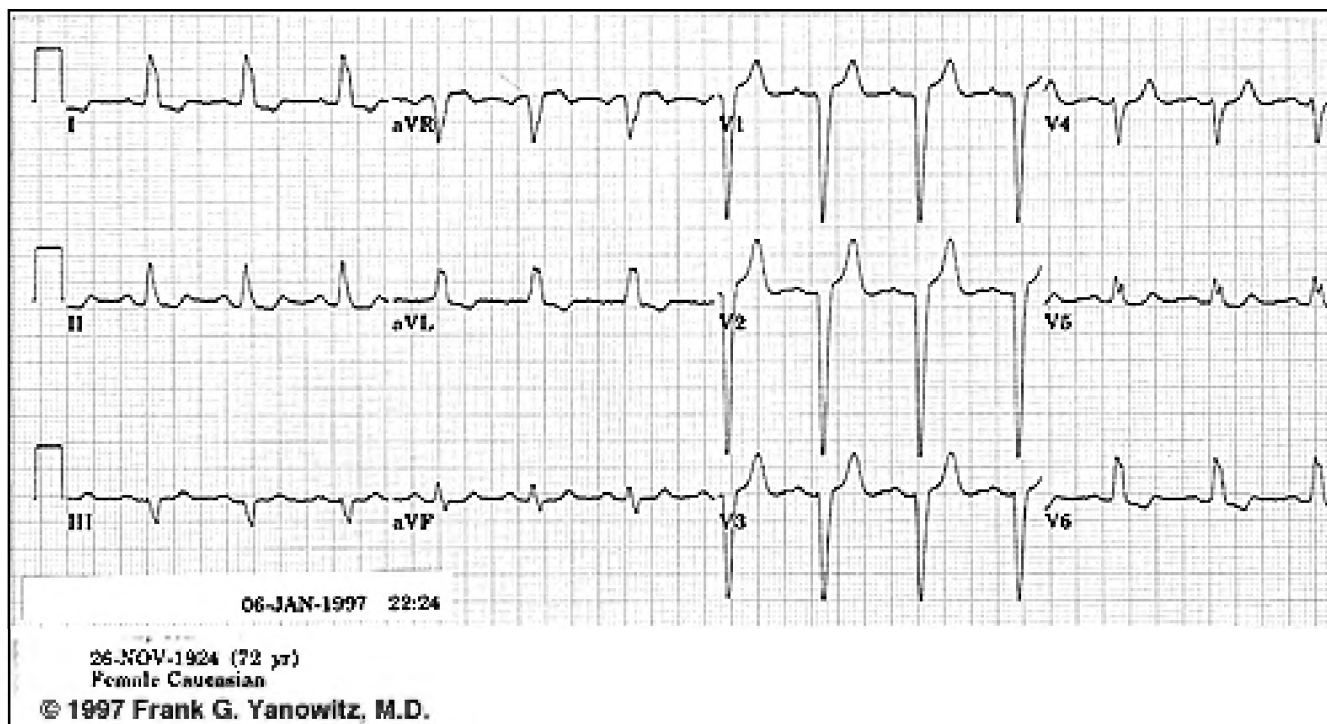
*Frank G. Yanowitz, M.D.*

LAFB is the most common of the intraventricular conduction defects. It is recognized by 1) left axis deviation; 2) rS complexes in II, III, aVF; and 3) small q in I and/or aVL.



## LAFB: Frontal Plane Leads-KH

*Frank G. Yanowitz, M.D.*

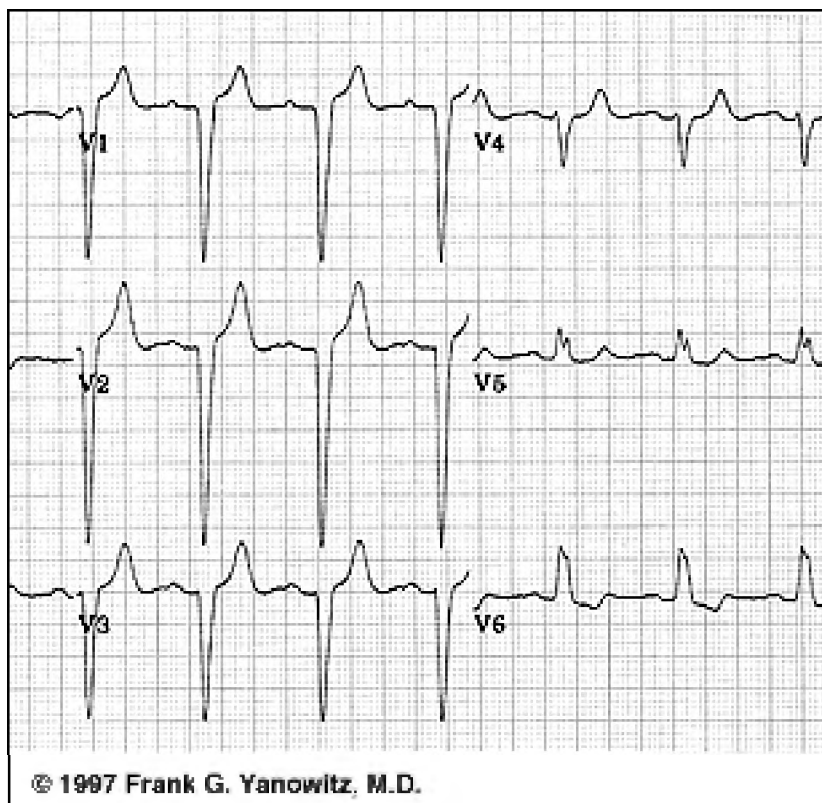


## Left Bundle Branch Block (LBBB)-KH

*Frank G. Yanowitz, M.D.*

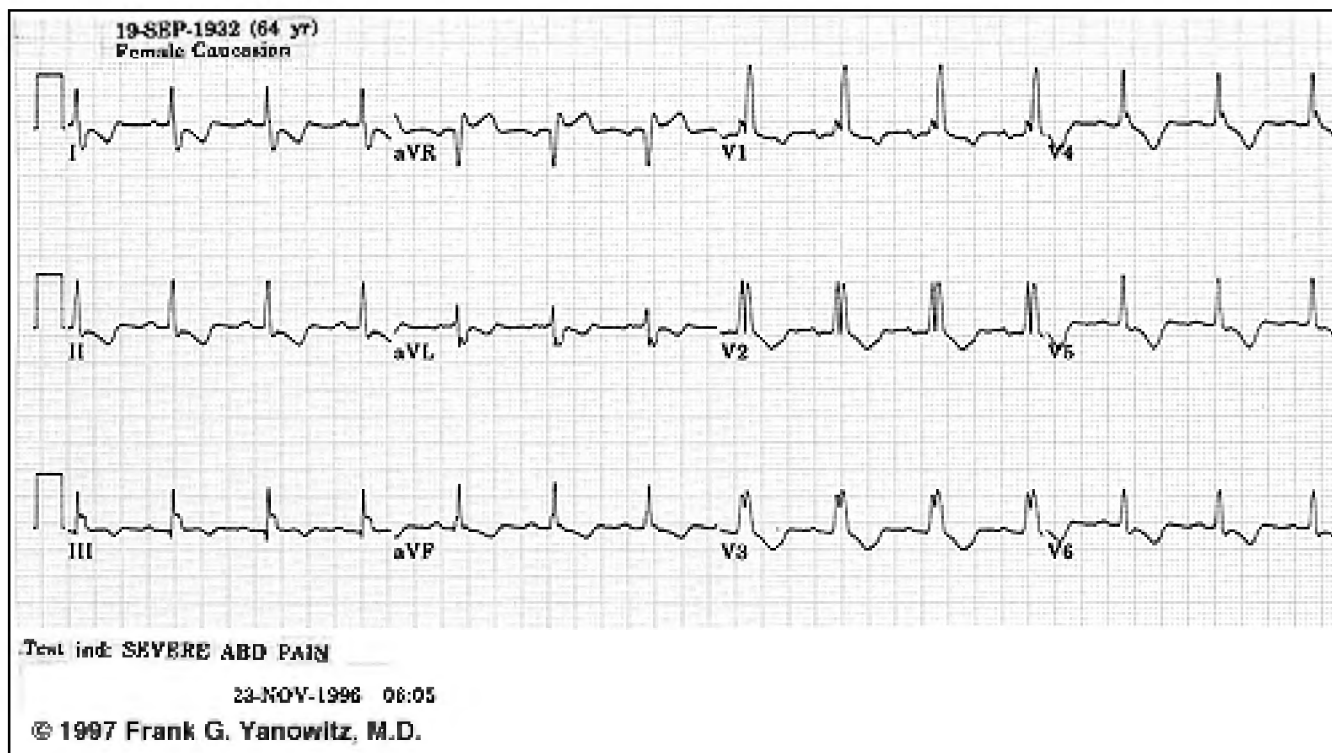
LBBB is recognized by 1) QRS duration  $>0.12s$ ; 2) monophasic R waves in I and V6; and 3) terminal QRS forces oriented leftwards and posterior. The ST-T waves should be oriented opposite to the terminal QRS forces.





## LBBB: Precordial Leads-KH

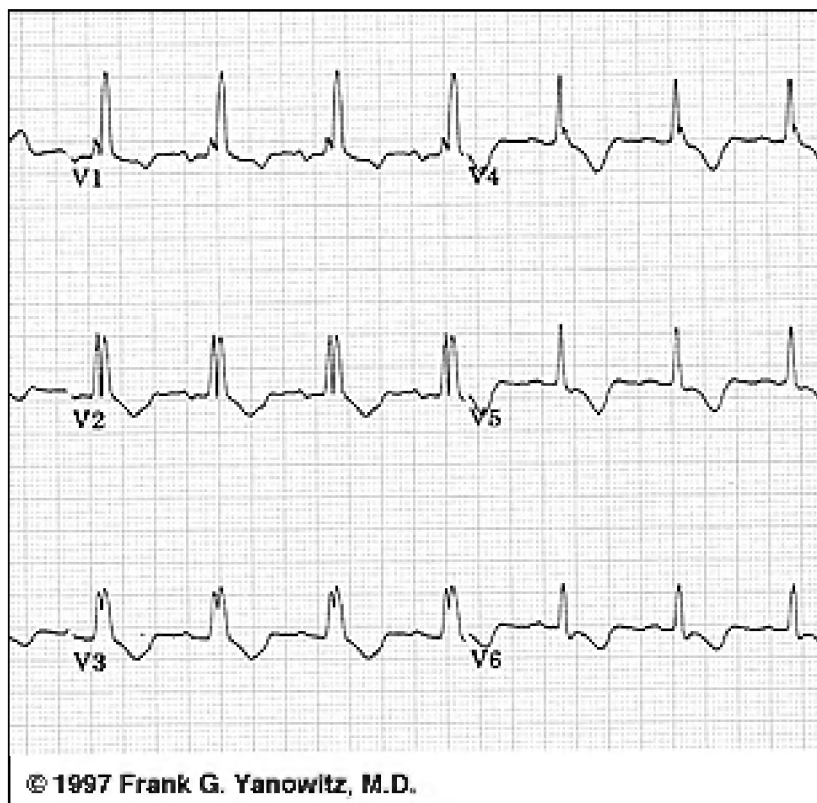
*Frank G. Yanowitz, M.D.*



## RBBB With Primary ST-T Wave Abnormalities-KH

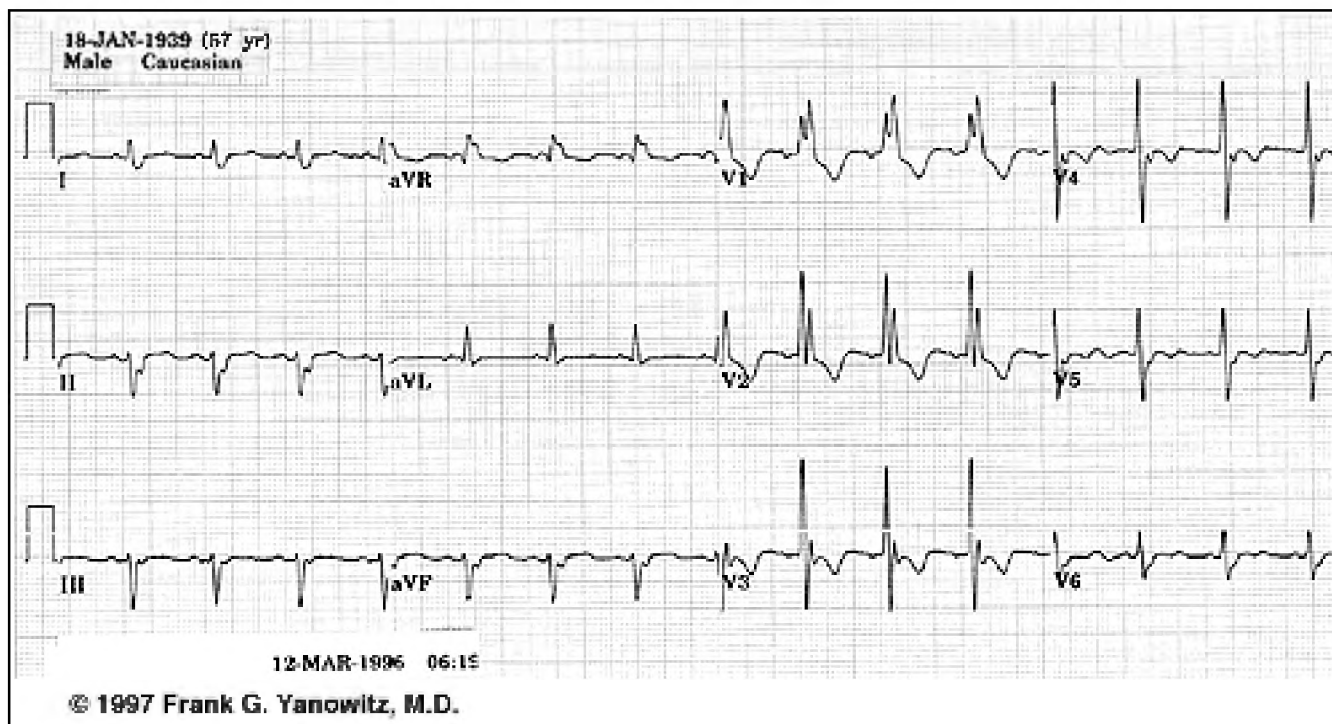
*Frank G. Yanowitz, M.D.*

RBBB is recognized by 1) rR' in V1; 2) QRS duration  $>0.12$ s; 3) terminal QRS forces oriented rightwards and anterior. In RBBB the ST-T waves should be oriented opposite to the terminal QRS forces. In this example there are "primary ST-T wave abnormalities" in leads I, II, aVL, V5, V6. In these leads the ST-T orientation is in the same direction as the terminal QRS forces.



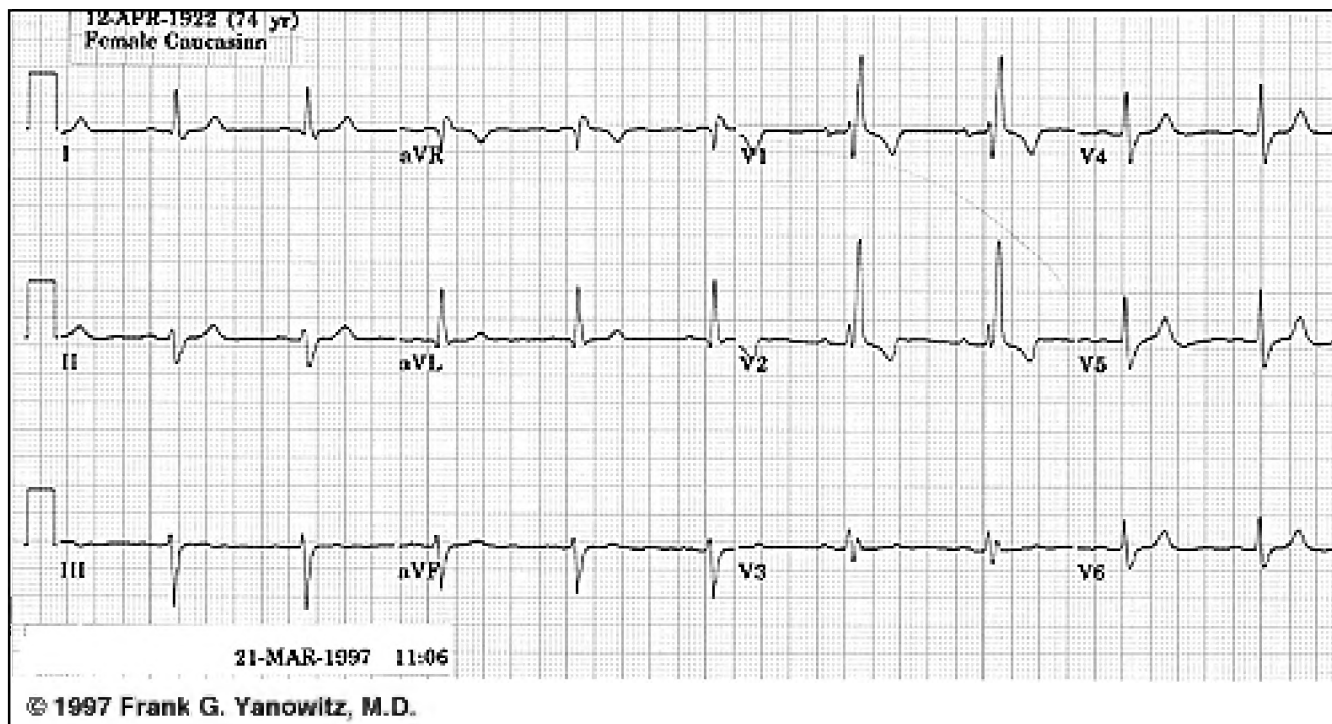
## RBBB with Primary ST-T Abnormalities: Precordial Leads-KH

*Frank G. Yanowitz, M.D.*



## Bifascicular Block: RBBB + LAFB-KH

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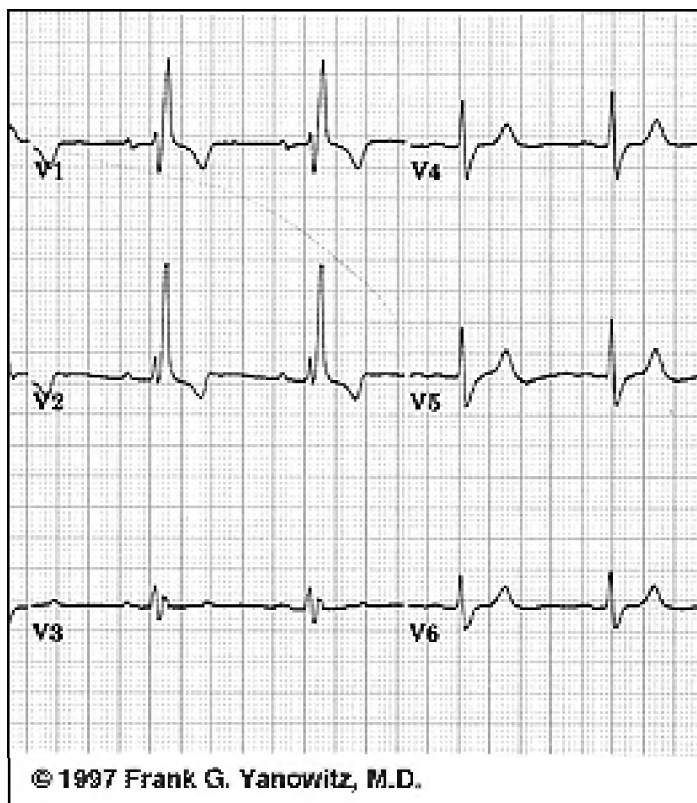


## Bifascicular Block: RBBB + LAFB-KH

*Frank G. Yanowitz, M.D.*

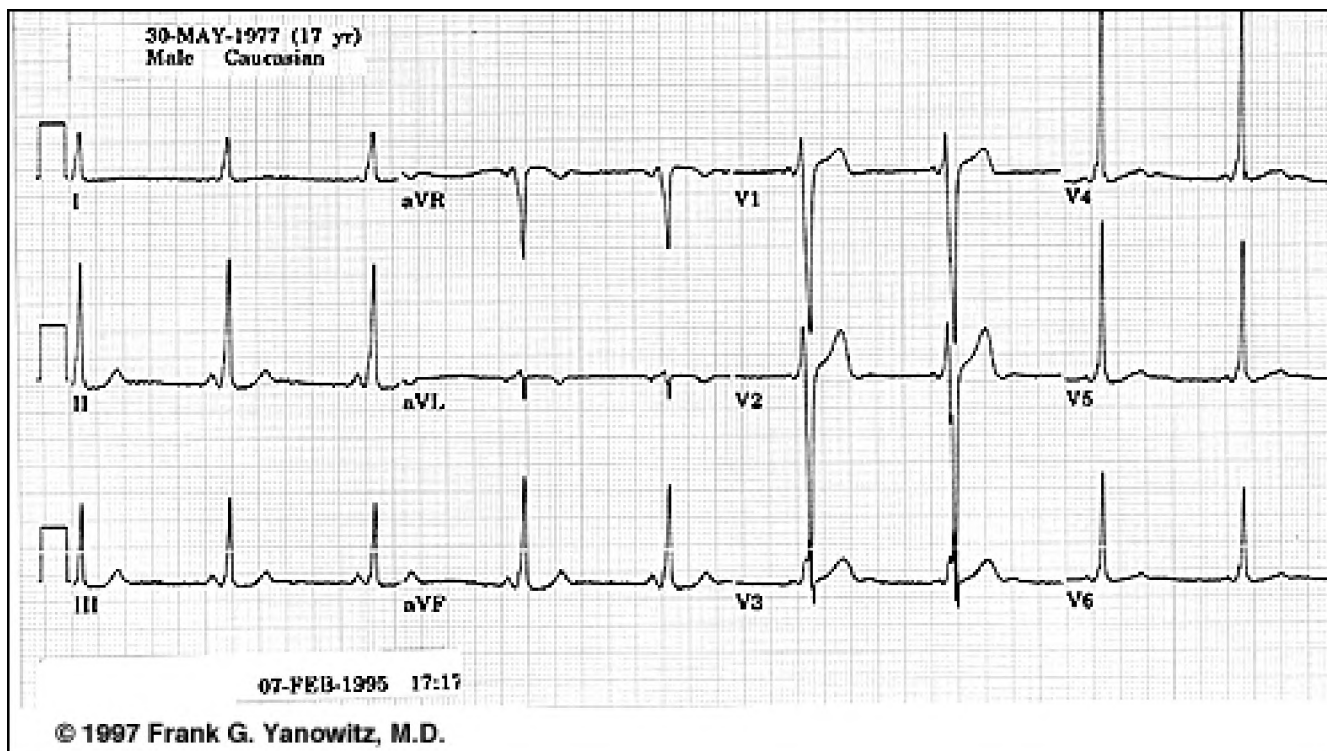
This is the most common of the bifascicular blocks. RBBB is most easily recognized in the precordial leads by the rSR' in V1 and the wide S wave in V6 (i.e., terminal QRS forces oriented rightwards and anterior). LAFB is best seen in the frontal plane leads as evidenced by left axis deviation (-50 degrees), rS complexes in II, III, aVF, and the small q in leads I and/or aVL.





## RBBB: Precordial Leads-KH

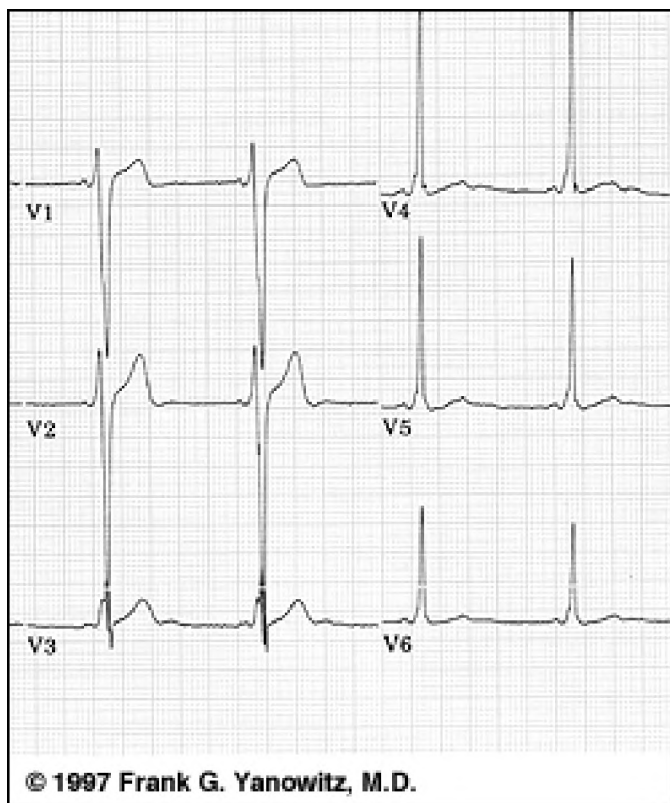
*Frank G. Yanowitz, M.D.*



## WPW Type Preexcitation-KH

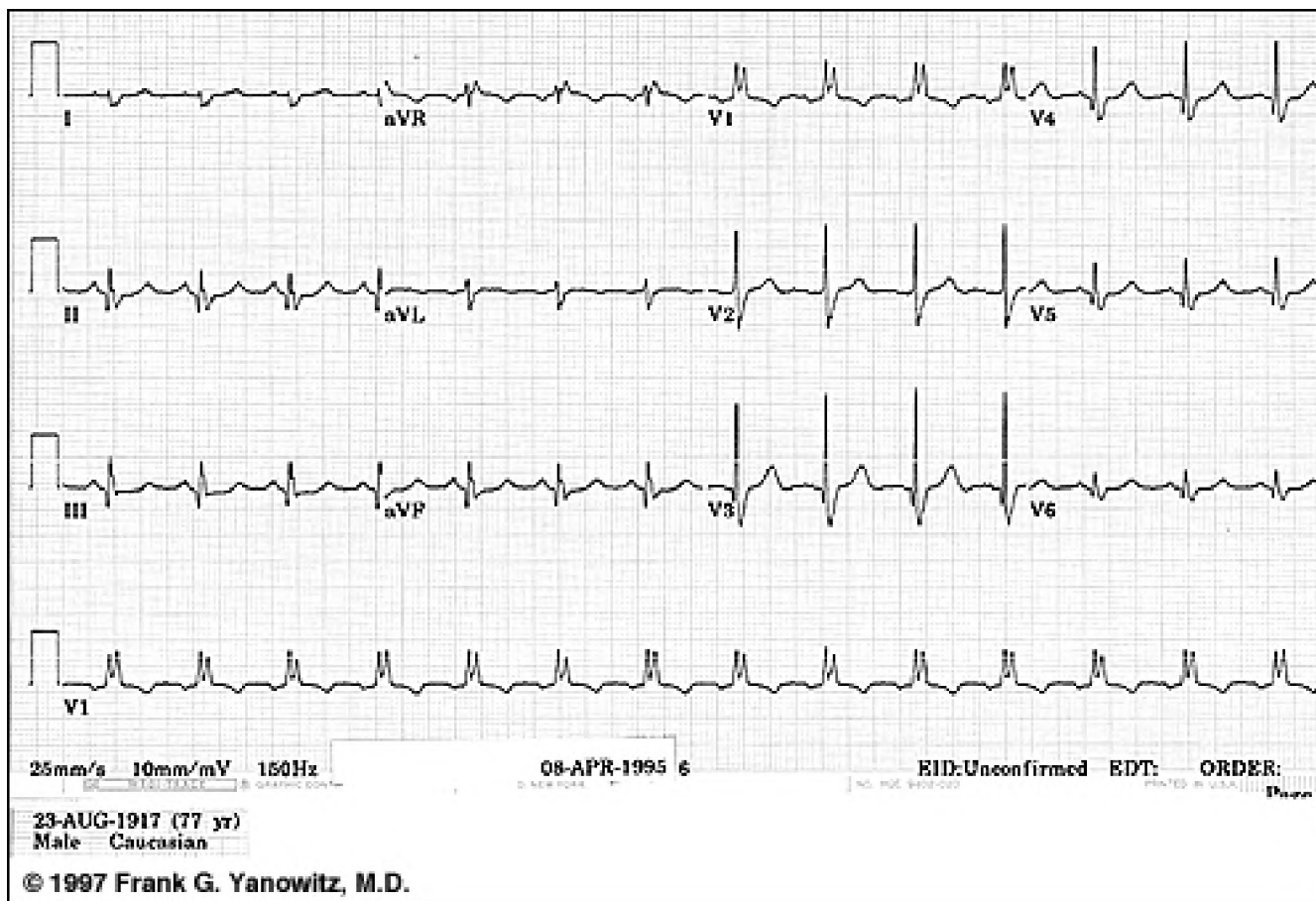
*Frank G. Yanowitz, M.D.*

Note the short PR and the subtle 'delta' wave at the beginning of the QRS complexes. The delta wave represents early activation of the ventricles in the region where the AV bypass tract inserts. The rest of the QRS is derived from the normal activation sequence using the bundle branches.



## WPW Type Preexcitation: Precordial Leads-KH

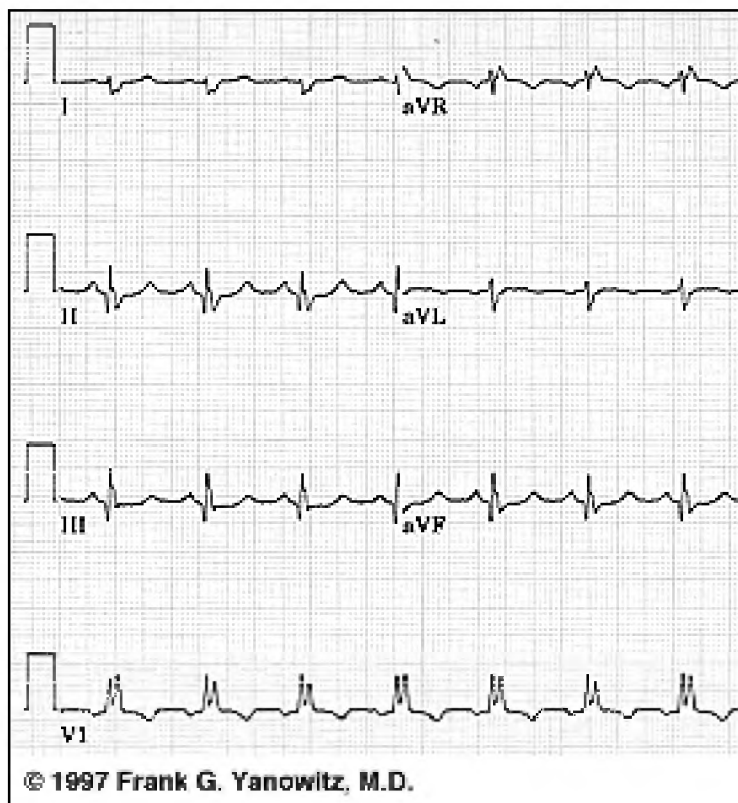
*Frank G. Yanowitz, M.D.*



## Infero-posterior MI & RBBB-KH

*Frank G. Yanowitz, M.D.*

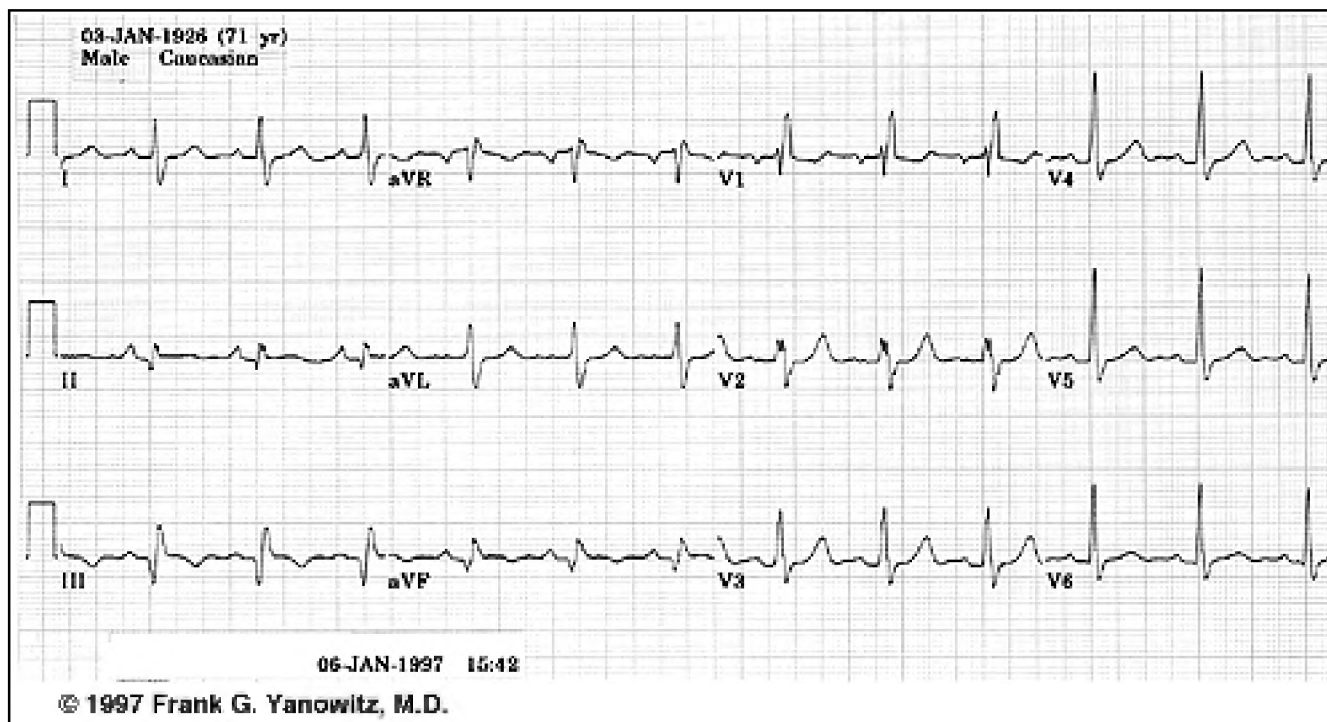
Deep Q waves in II, III, aVF plus tall R waves in V1-2 are evidence for this infero-posterior MI. The wide QRS ( $>0.12$ s) and RR' complex in V1 are evidence for RBBB. Any time RBBB has an initial R in V1 equal to or greater than the R', true posterior MI must be considered. Q waves in V5-6 suggest an apical lateral wall extension of this large MI.



## Infero-posterior MI & RBBB: Frontal Plane Leads + V1-KH

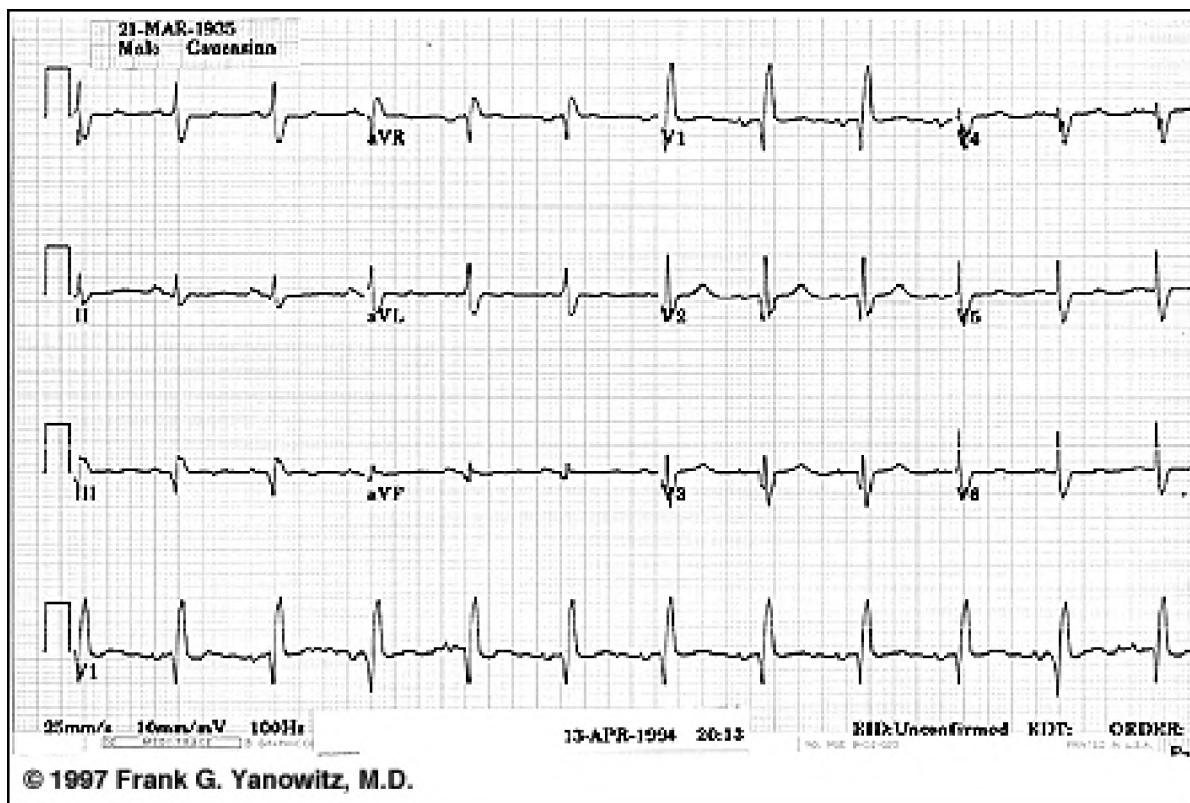
*Frank G. Yanowitz, M.D.*





## Inferior MI and RBBB-KH

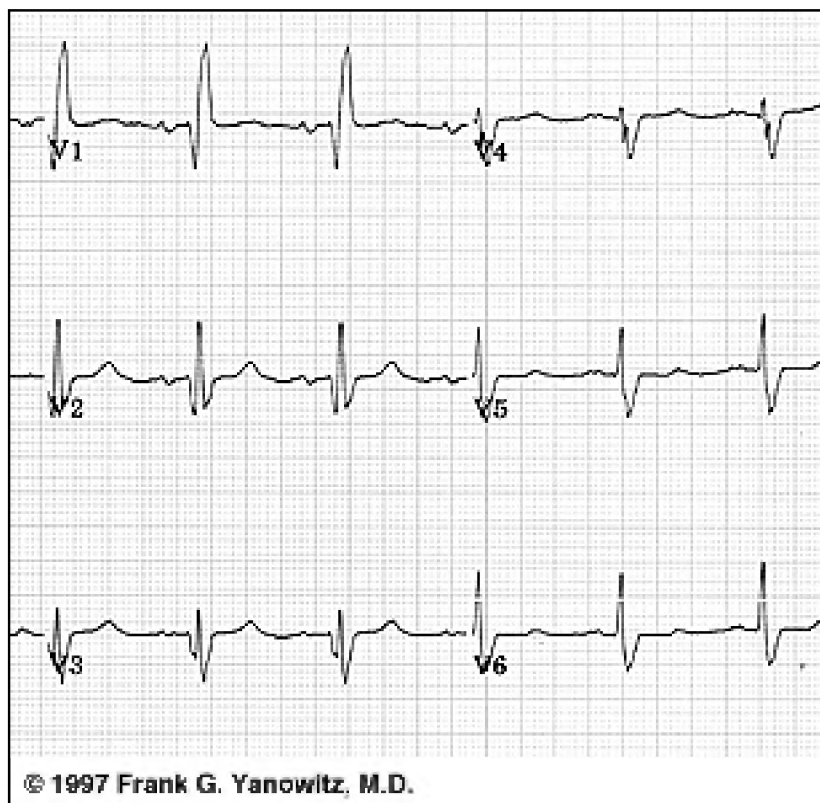
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## Inferior & Anteroseptal MI + RBBB-KH

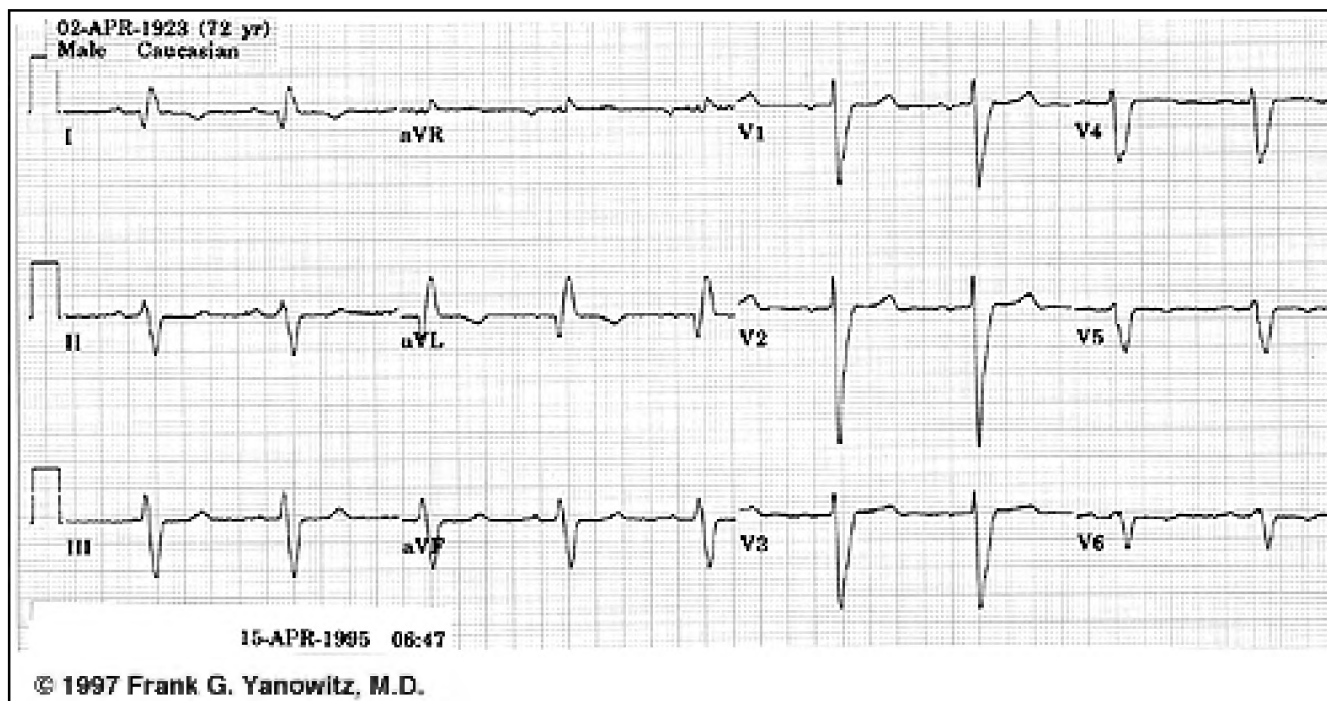
*Frank G. Yanowitz, M.D.*

Pathologic Q waves are seen in leads II, III, aVF (inferior MI) and in leads V1-3 (anteroseptal MI). RBBB is recognized by the wide QRS ( $>0.12$ s) and the anterior/rightwards orientation of terminal QRS forces. When an anteroseptal MI complicates RBBB (or visa versa) the rSR' complex in V1 (typical of RBBB) becomes a qR complex.



## Anteroseptal MI With RBBB: Precordial Leads-KH

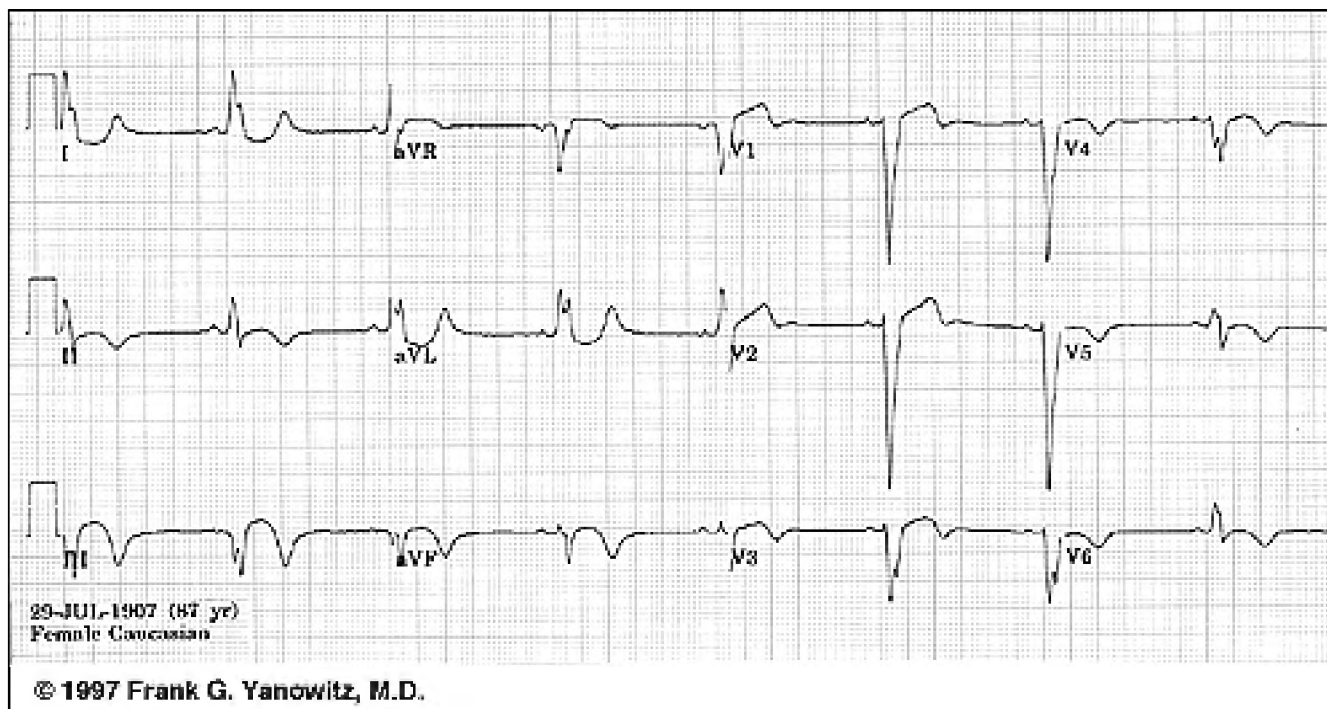
*Frank G. Yanowitz, M.D.*



## Atypical LBBB with Q Waves in Leads I and aVL-KH

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In typical LBBB, there are no initial Q waves in leads I, aVL, and V6. If Q waves are present in 2 or more of these leads, myocardial infarction is present.

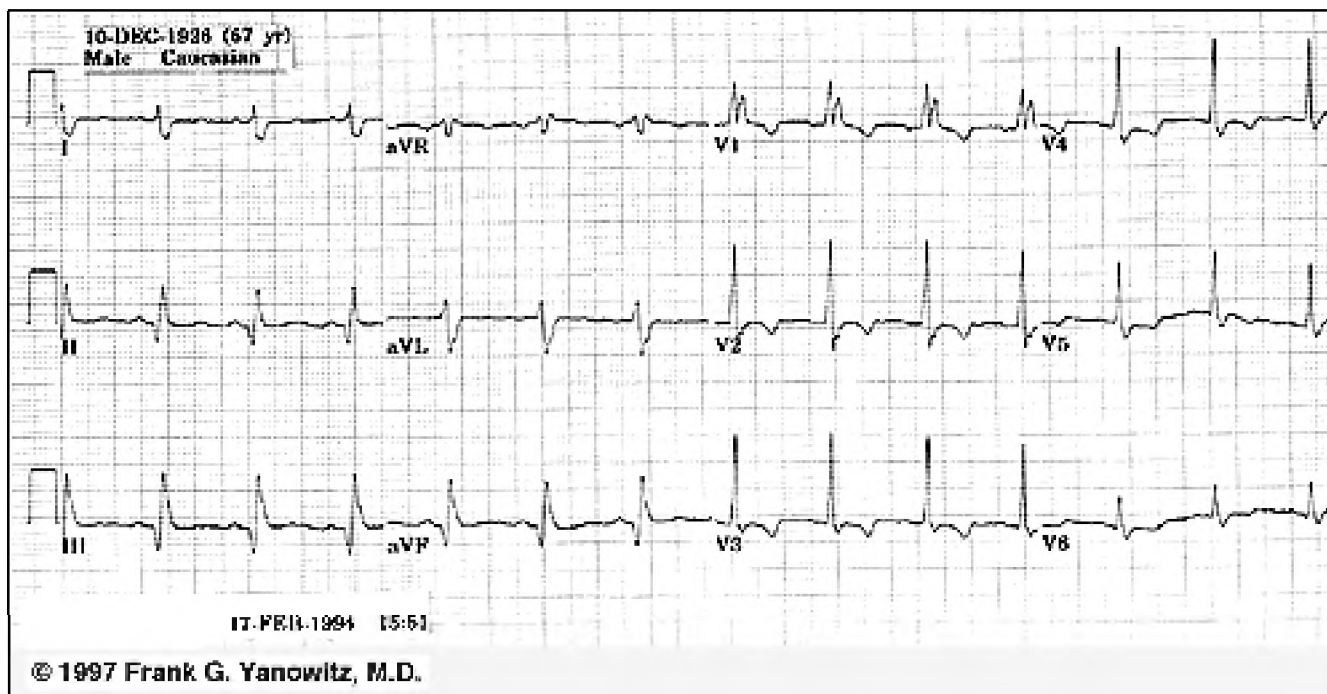


## Atypical LBBB with Primary T Wave Abnormalities-KH

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Primary T wave abnormalities in LBBB refer to T waves in the same direction as the major deflection of the QRS. These are seen in leads I, III, aVL, V2-4. Most likely diagnosis is myocardial infarction.

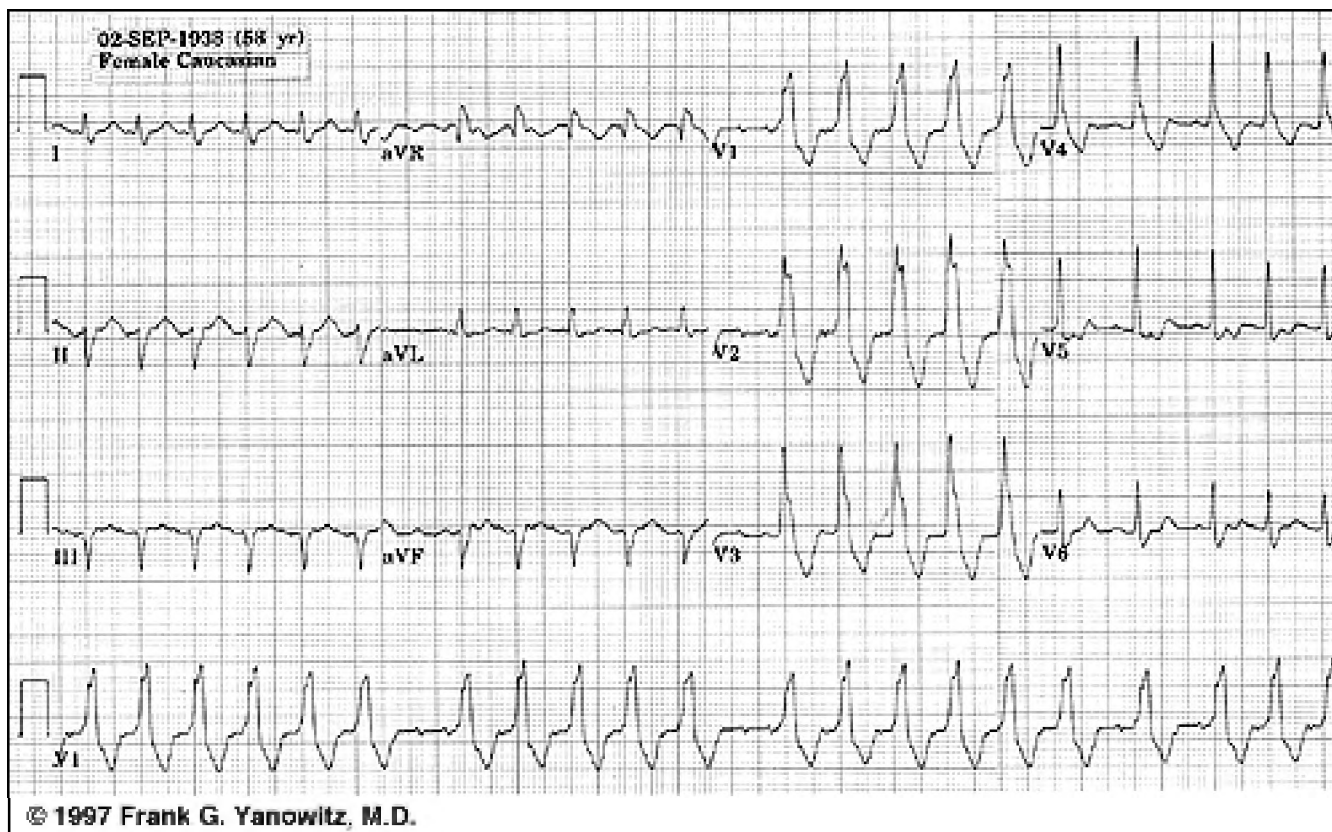




## Infero-posterior MI with RBBB-KH

*Frank G. Yanowitz, M.D., copyright 1997*

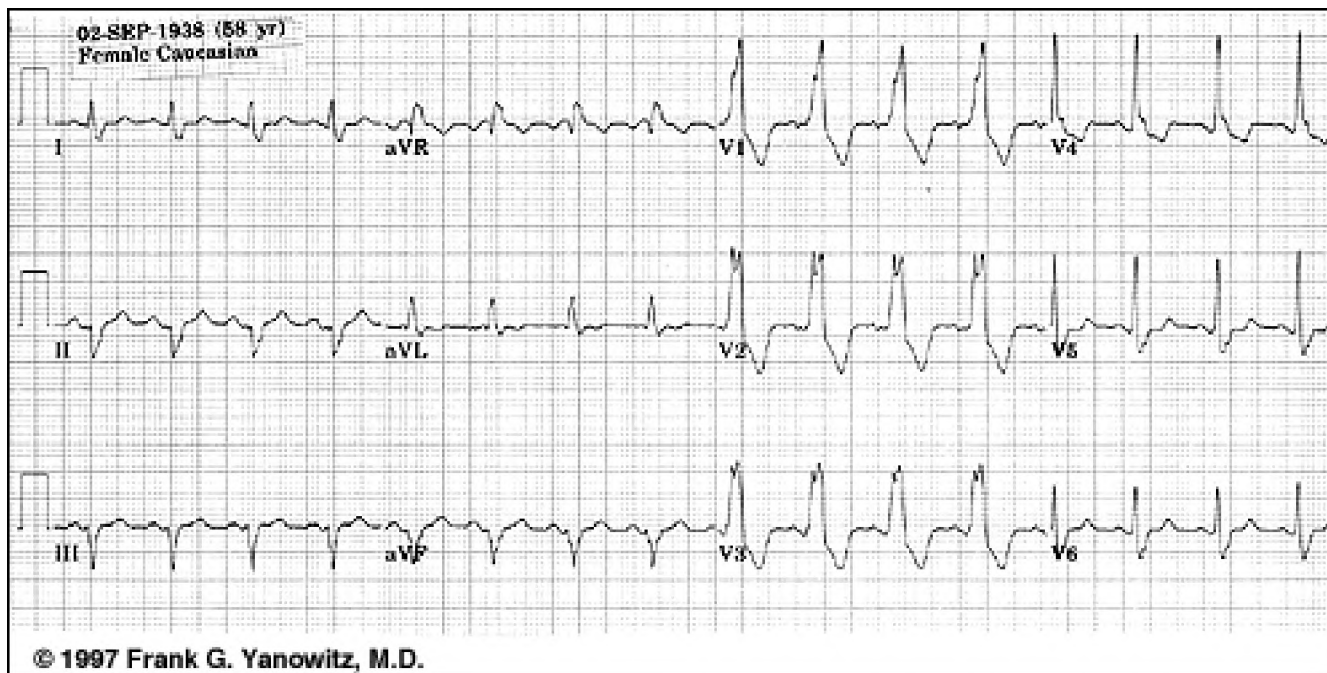
This is an unusual RBBB because the initial R wave is taller than the R' wave in lead V1. This is the clue for true posterior MI. The tall initial R wave in V1 is a "pathologic R" wave analagous to the "pathologic Q" wave of an anterior MI.



## RBBB + LAFB = Bifascicular block-KH

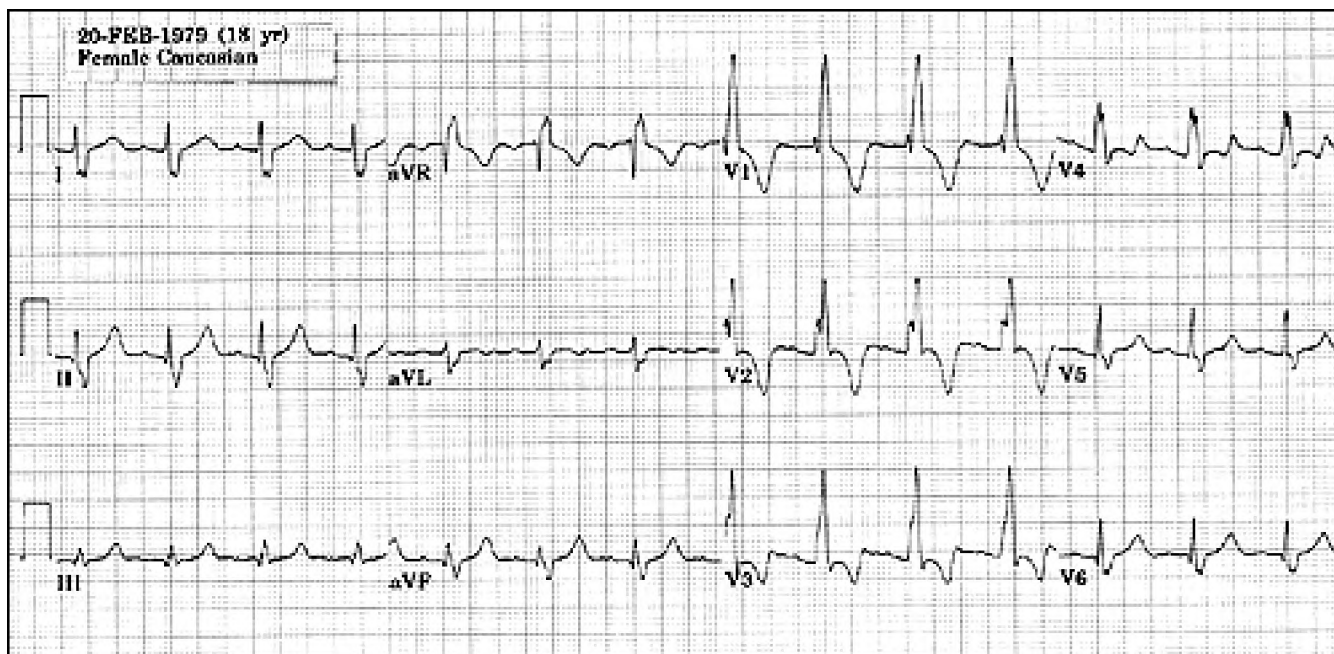
*Frank G. Yanowitz, M.D. copyright 1997*

The RBBB is diagnosed by the wide QRS with prominent anterior (e.g., V1) and late rightward (e.g., I, V6) forces. The LAFB is recognized by the marked left axis deviation (-75 degrees) in the frontal plane, rS complexes in II, III, aVF, and the tiny q-wave in aVL.



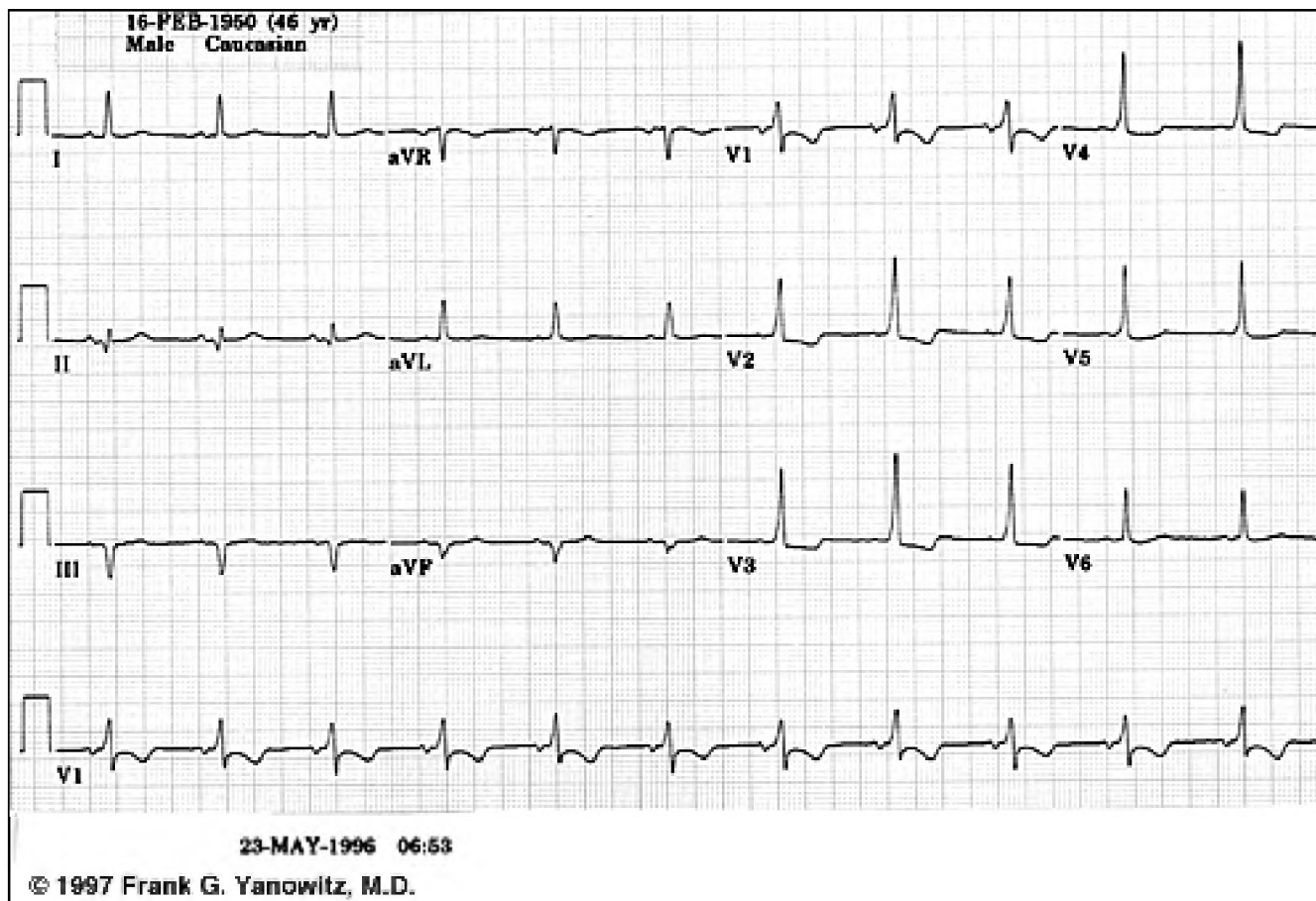
## RBBB + LAFB: Bifascicular Block-KH

*Frank G. Yanowitz, M.D. copyright 1997*



## Right Bundle Branch Block (RBBB)-KH

*Frank G. Yanowitz, M.D. copyright 1997*

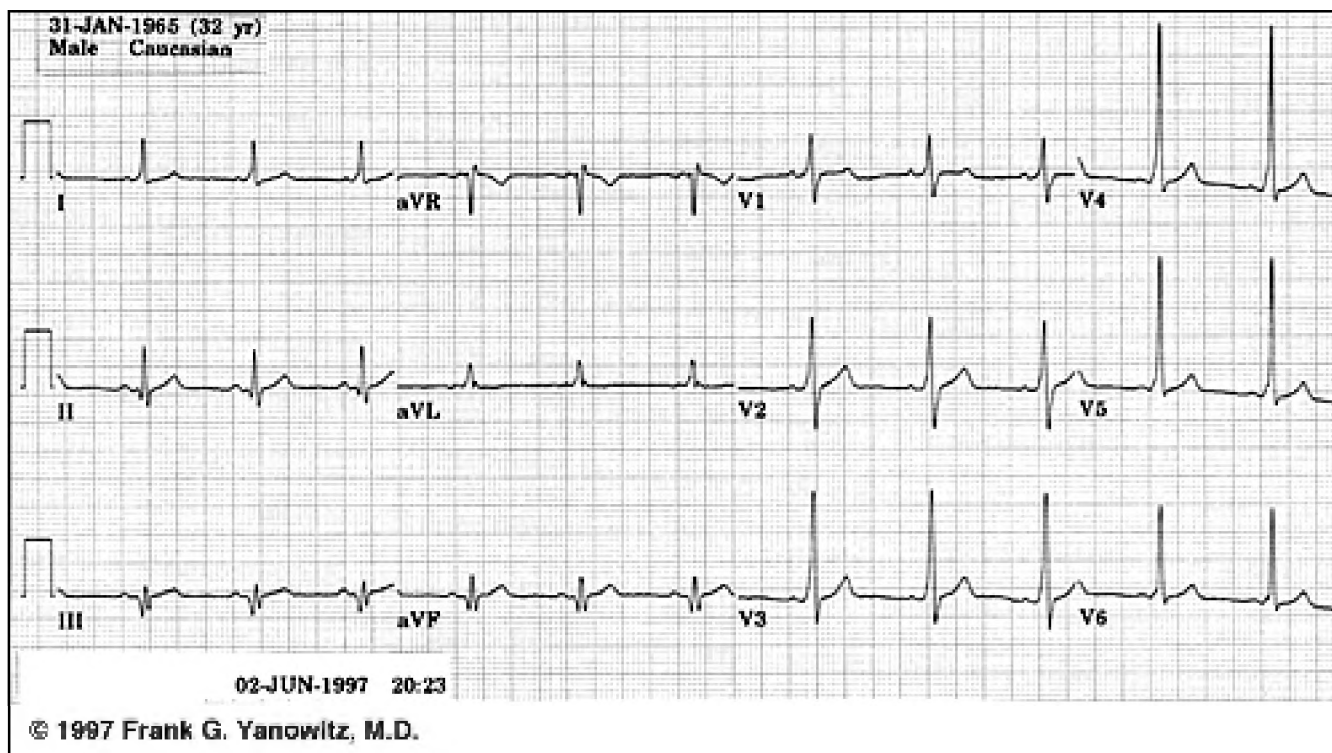


## WPW and Pseudo-inferior MI

*Frank G. Yanowitz, M.D. Copyright 1998*

Short PR intervals and delta waves are best seen in leads V1-5. Pseudo-Q waves, seen in leads II, III, and aVF, are actually negative delta waves. There is no inferior MI on this ECG.



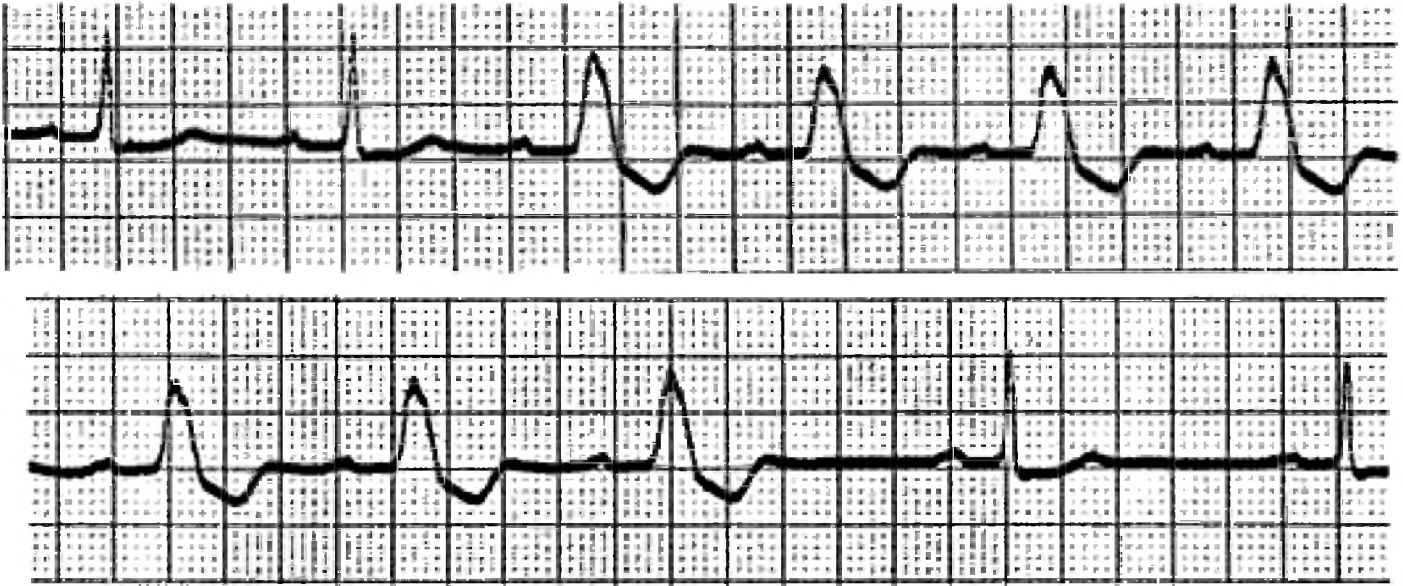


## WPW with a Pseudo-inferior MI

*Frank G. Yanowitz, M.D. Copyright 1998*

The short PR intervals and delta waves are best seen in the precordial leads. "Q" waves in leads II, III, aVF are actually negative delta waves and not indicative of an old inferior MI.

## Lead I

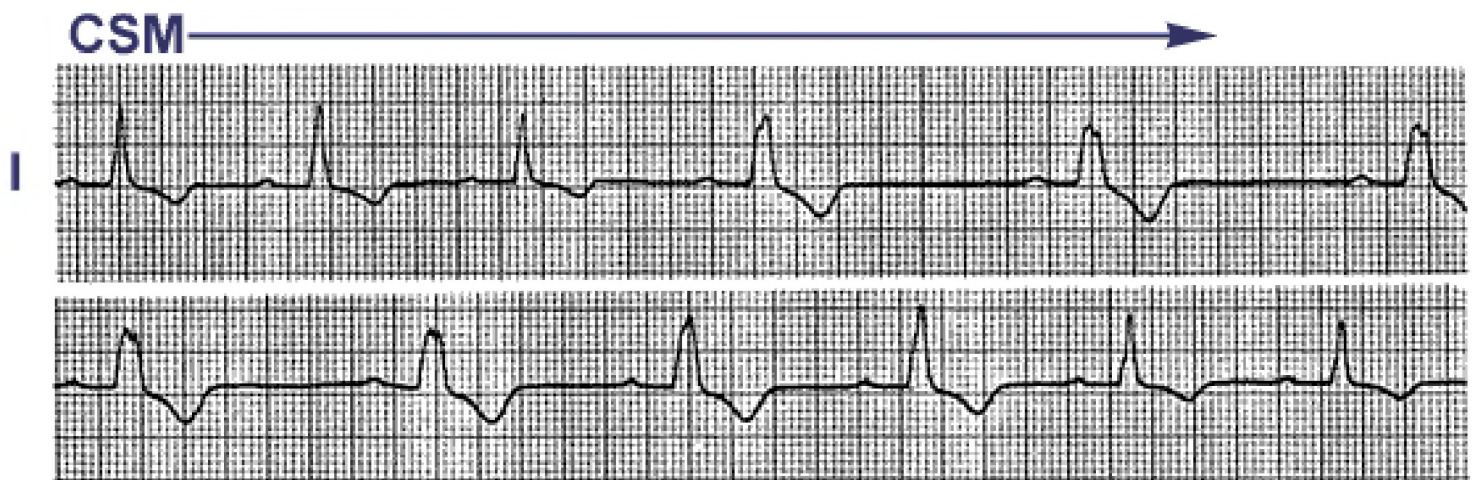


## Rate-dependent LBBB

## Rate-dependent LBBB-KH

*Frank Yanowitz Copyright 1996*

In this rhythm strip of sinus arrhythmia, the faster rates have a LBBB morphology. In some patients with a diseased left bundle branch, the onset of LBBB usually occurs initially as a rate-dependent block; i.e., the left bundle fails to conduct at the faster rate because of prolonged refractoriness.

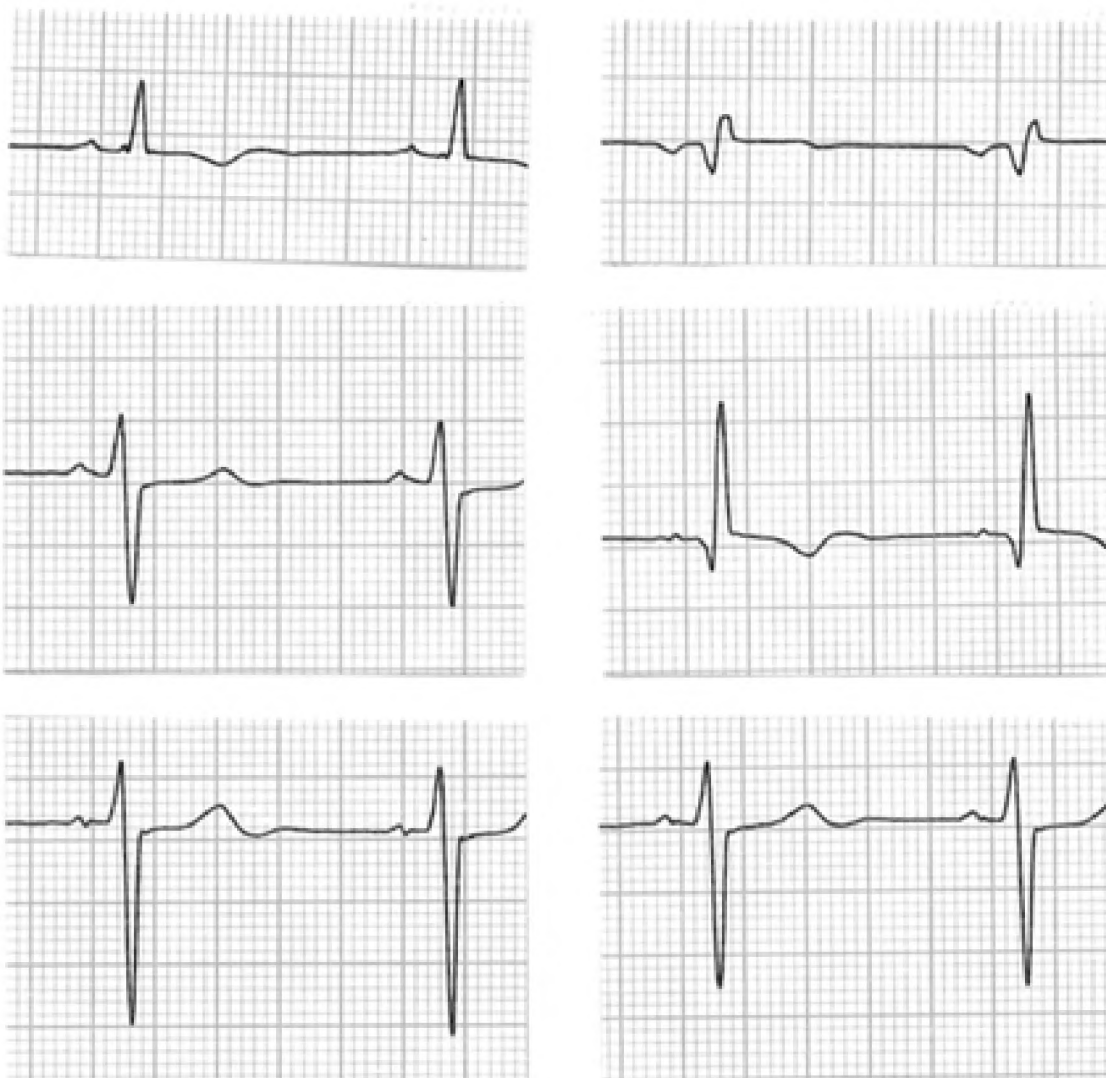


**Bradycardia-dependent LBBB (phase 4 block)**  
**CSM = carotid sinus massage**

## **Bradycardia-dependent LBBB With Carotid Sinus Massage-KH**

*Frank Yanowitz Copyright 1996*

When carotid sinus massage slows the heart rate in this example, the QRS widens into a LBBB. This form of rate-dependent bundle branch block is thought to be due to latent pacemakers in the bundle undergoing phase 4 depolarization; when the sinus impulse enters the partially depolarized bundle, slowed conduction or heart block occurs in that bundle branch.



## Left Anterior Fascicular Block: Frontal Plane Leads-KH

*Frank Yanowitz Copyright 1996*

Left anterior fascicular block, LAFB, is recognized by left axis deviation of  $-45$  degrees or greater; rS complexes in II, III, aVF; and a small Q wave in I and/or aVL.





## Right Bundle Branch Block



P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	.12 to .20	>.12	RSR' in V1

## Right Bundle Branch Block-KH

*Frank G. Yanowitz, M.D., copyright 1997*

## Left Bundle Branch Block



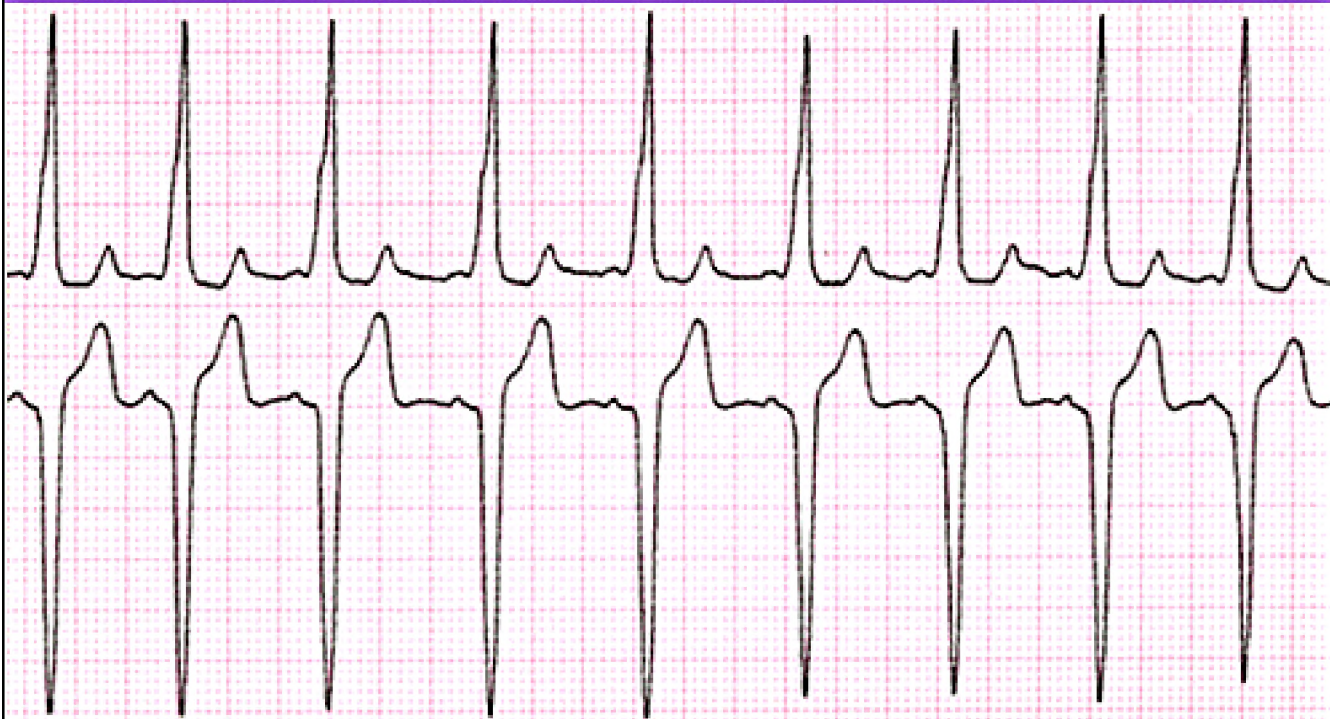
P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	.12 to .20	$\geq .12$	RR' in V5

## Left Bundle Branch Block - Marquette-KH

*Marquette Electronics Copyright 1996*



## Preexcitation Syndrome



P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	<.12	Usually >.10	Delta wave distorts QRS

## WPW Type Preexcitation - Marquette-KH

*Marquette Electronics Copyright 1996*

## Right Bundle Branch Block

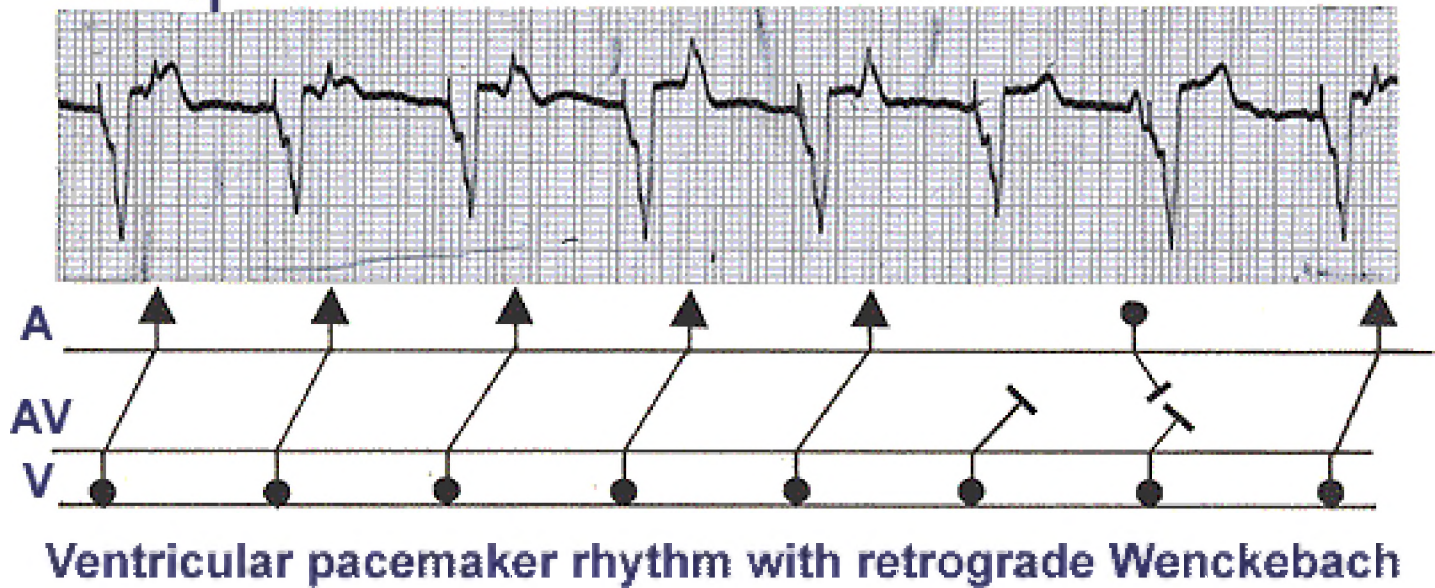


P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	.12 to .20	>.12	RSR' in V1

## RBBB - Marquette-KH

*Marquette Electronics Copyright 1996*



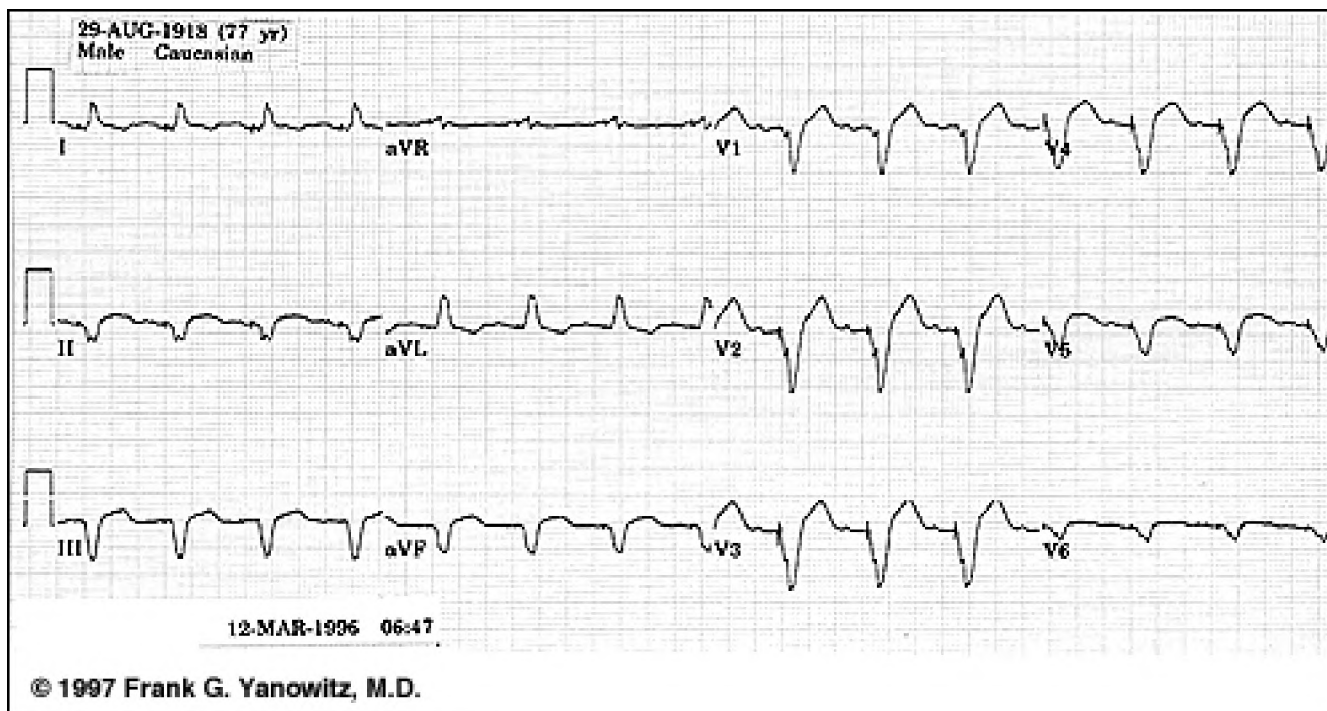
Lead V<sub>1</sub>

## Ventricular Paced Rhythm With Retrograde Wenckebach-KH

*Frank Yanowitz Copyright 1996*

Retrograde atrial captures from a ventricular paced rhythm are occurring with increasing R-P intervals; i.e., retrograde Wenckebach. The ladder diagram indicates that after the blocked retrograde event, a single sinus P wave is seen dissociated from the ventricular rhythm.





## Ventricular Pacemaker Rhythm-KH

*Frank G. Yanowitz, M.D.*

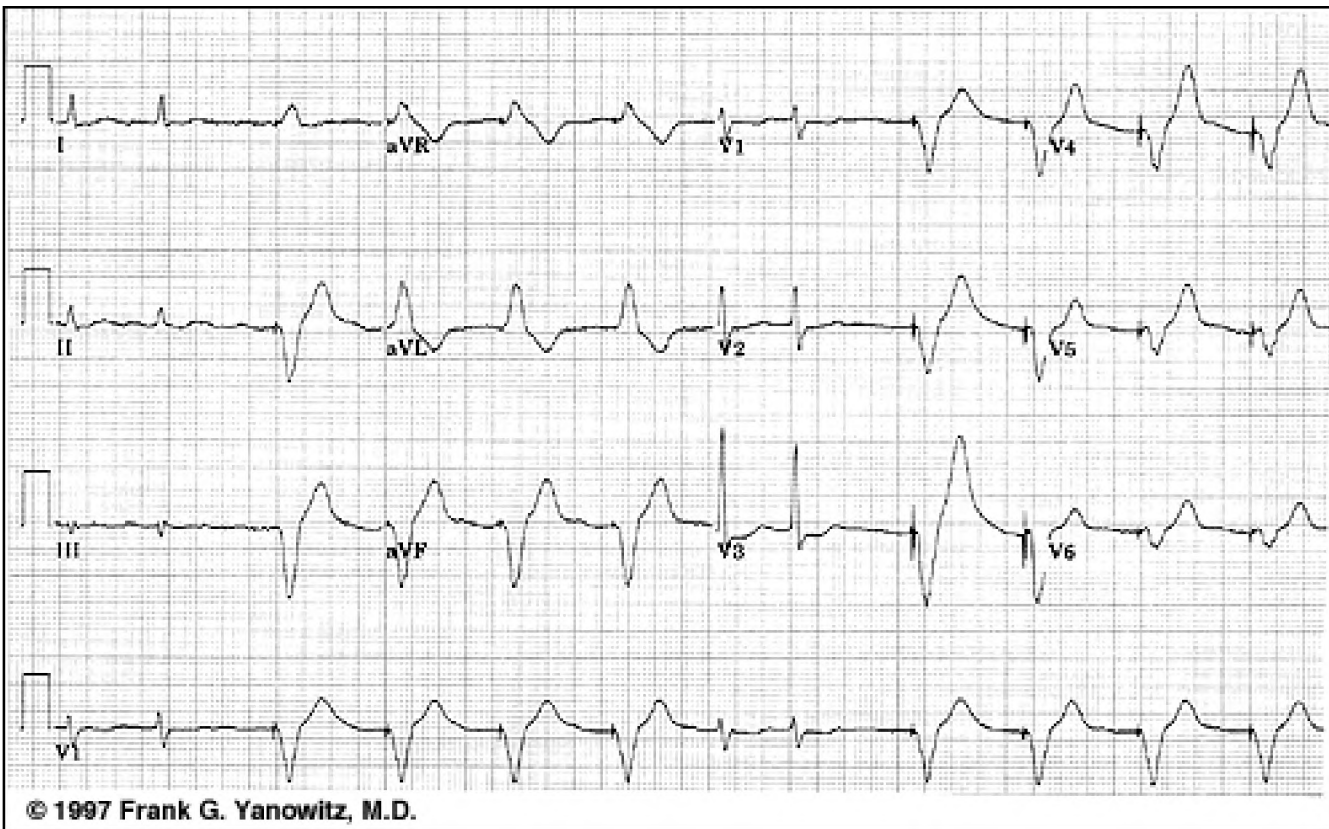
Note the small pacemaker spikes before the QRS complexes in many of the leads. In addition, the QRS complex in V1 exhibits ventricular ectopic morphology; i.e., there is a slur or notch at the beginning of the S wave, and  $>60$ ms delay from onset to QRS to nadir of S wave. This rules against a supraventricular rhythm with LBBB.



## Ventricular Pacemaker Rhythm: V1-3-KH

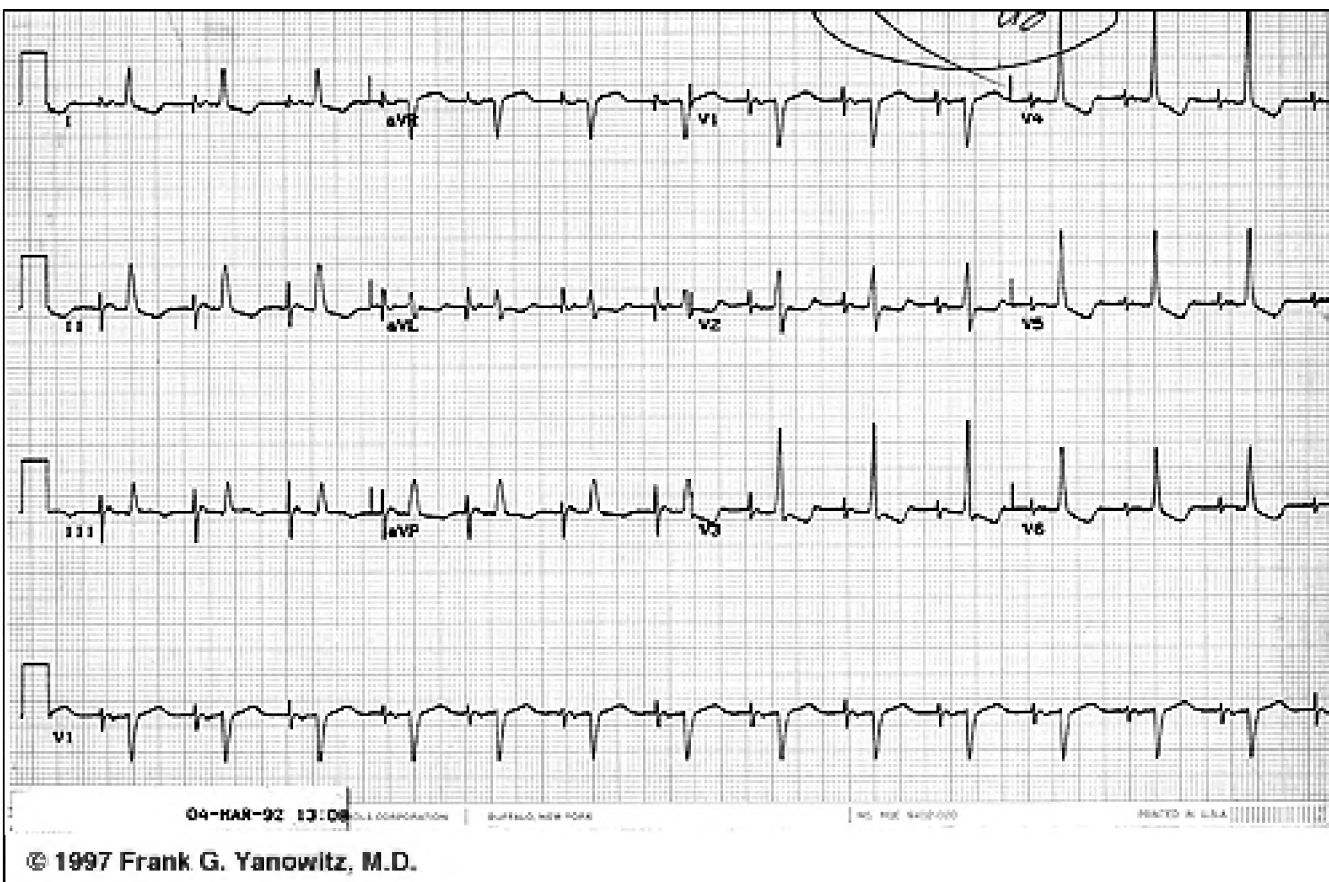
*Frank G. Yanowitz, M.D.*

Note the small pacemaker spikes before the QRS complexes. In addition, the QRS complex in V1-3 exhibits ventricular ectopic morphology; i.e., there is a slur or notch at the beginning of the S wave, and >60ms delay from onset to QRS to nadir of S wave. This rules against a supraventricular rhythm with LBBB.



## Ventricular Pacemaker: Demand mode functioning-KH

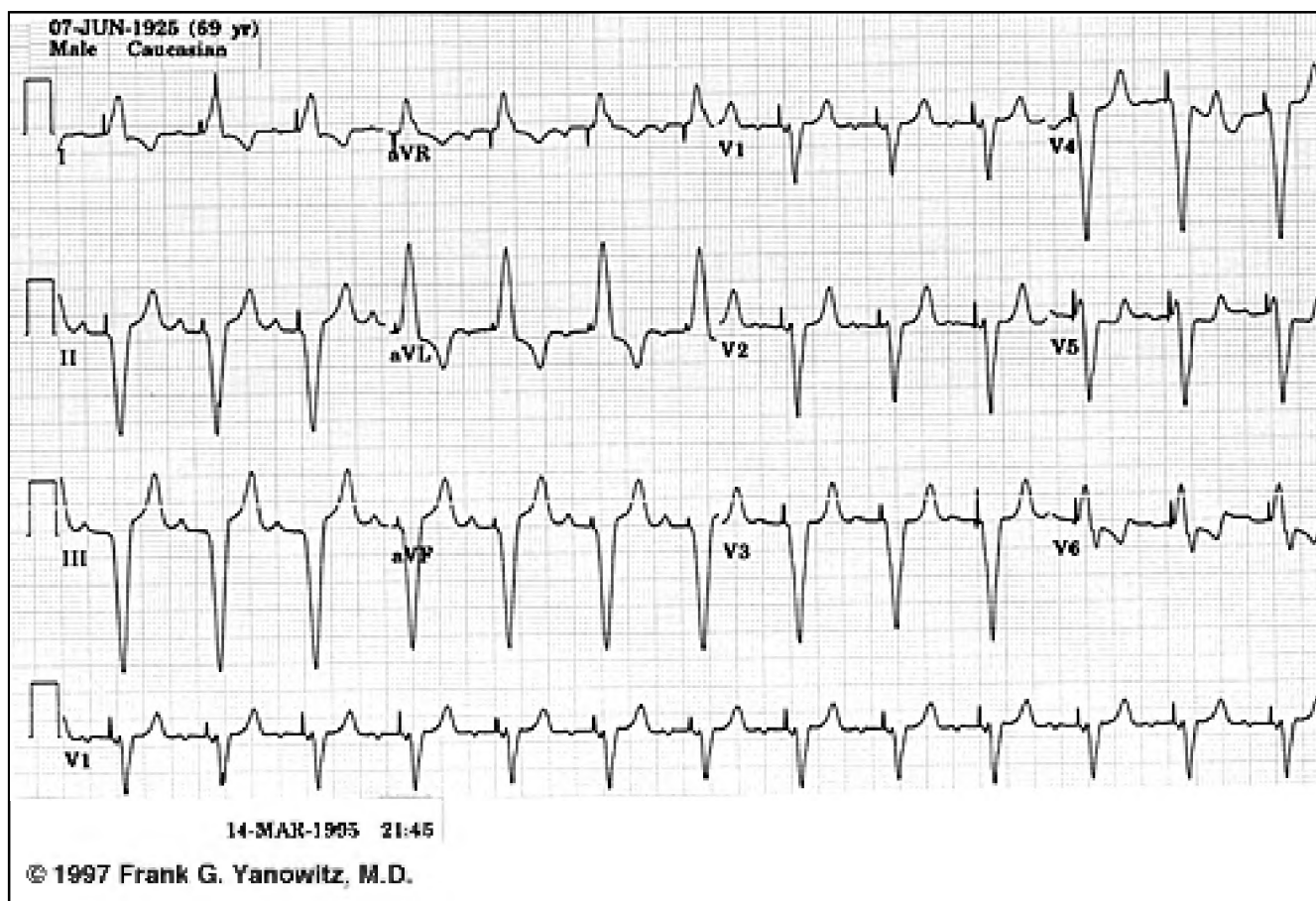
*Frank G. Yanowitz, M.D. copyright 1997*



## Atrial Pacemaker Rhythm

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Pacemaker spikes are seen before each QRS complex and initiate a tiny P wave. Diffuse ST-T wave abnormalities are present as well as prominent anterior forces ( $R > S$  in lead V2). The cause of these abnormalities is unknown.

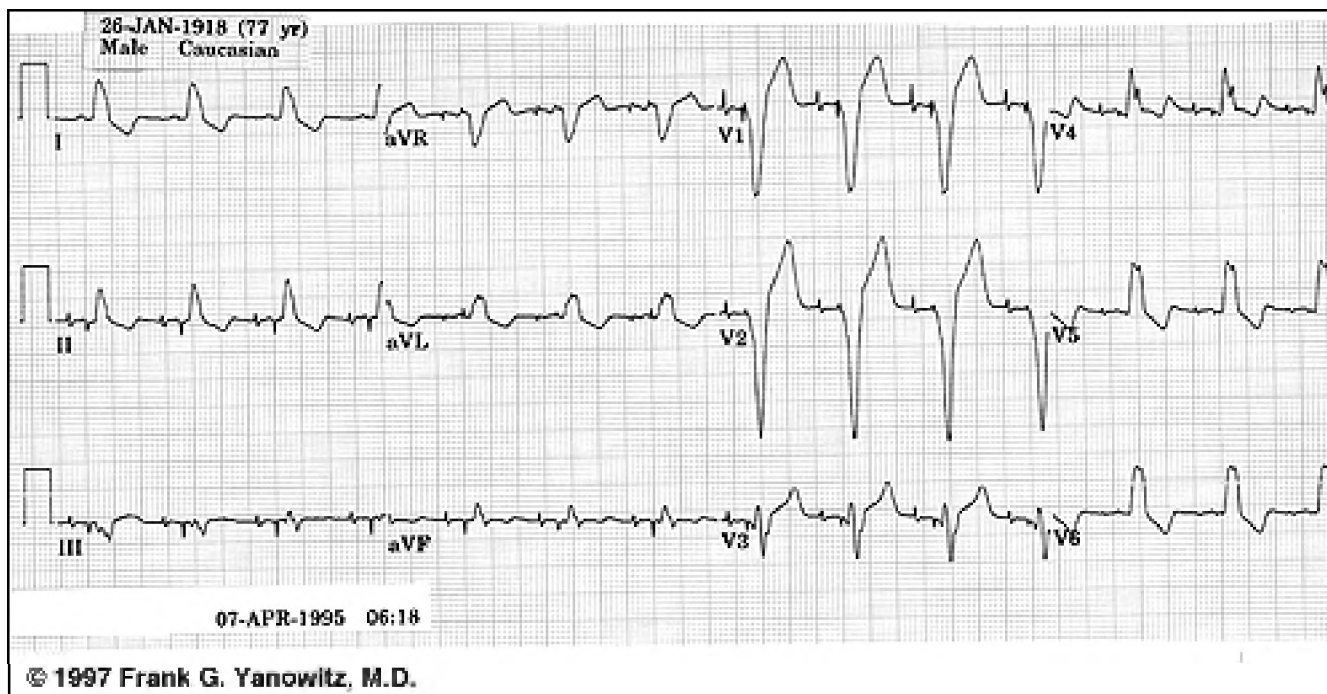


## AV Sequential Pacing

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Pacemaker spikes immediately precede each QRS complex indicating ventricular pacing. Each QRS also has a preceding sinus P wave indicating that the patient is in sinus rhythm. An atrial pacing wire senses the sinus rhythm and coordinates ventricular pacing to allow atrial contraction to contribute to ventricular filling. This is a common form of dual chamber pacing.





## AV Sequential Pacing

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In this ECG both atria and ventricles are being paced. Two pacemaker spikes are seen before each QRS, one for the atria and one for the ventricles (best seen in lead V1).

## Atrial Pacemaker (single chamber)



**One spike producing an abnormal P wave (atrial capture) followed by a normal QRS**

## Electronic Atrial Pacing - Marquette-KH

*Marquette Electronics Copyright 1996*

## AV Sequential Pacemaker (dual chamber)

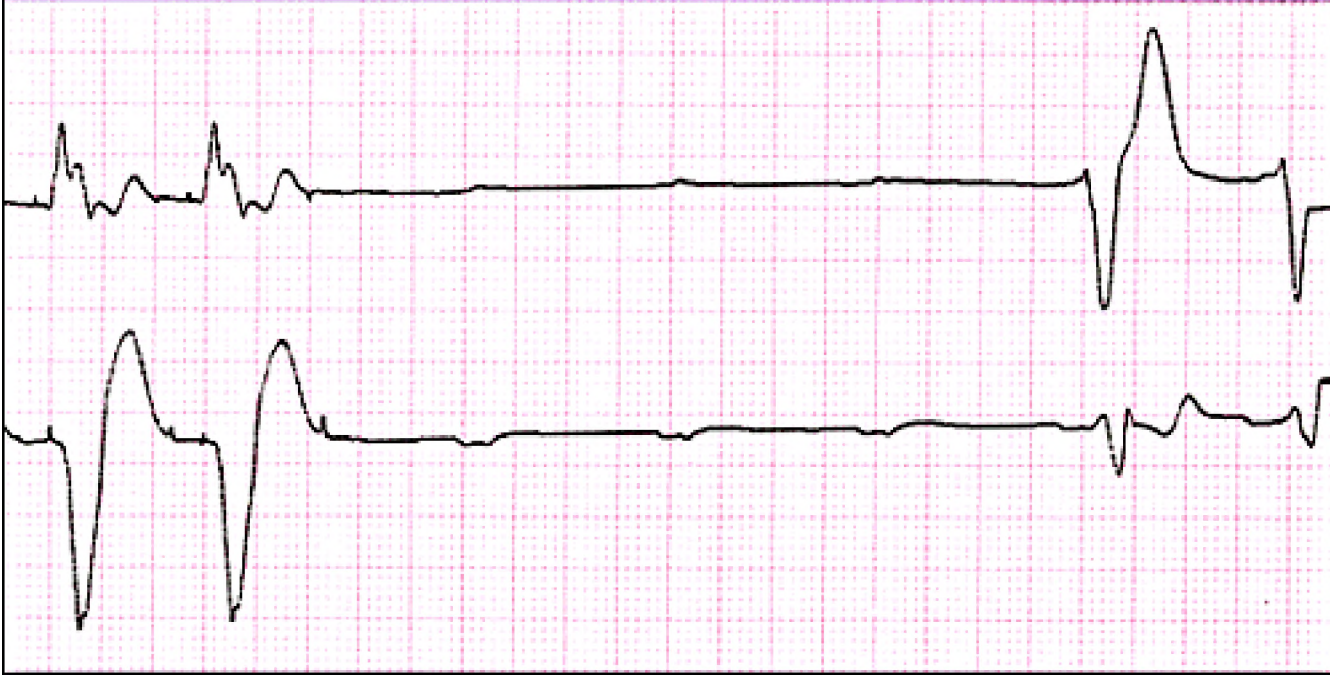


**One spike followed by an abnormal P (atrial capture) followed by a  
Second spike producing a wide QRS (ventricular capture).**

## AV Sequential Pacemaker - Marquette-KH

*Marquette Electronics Copyright 1996*

## Failure to Output



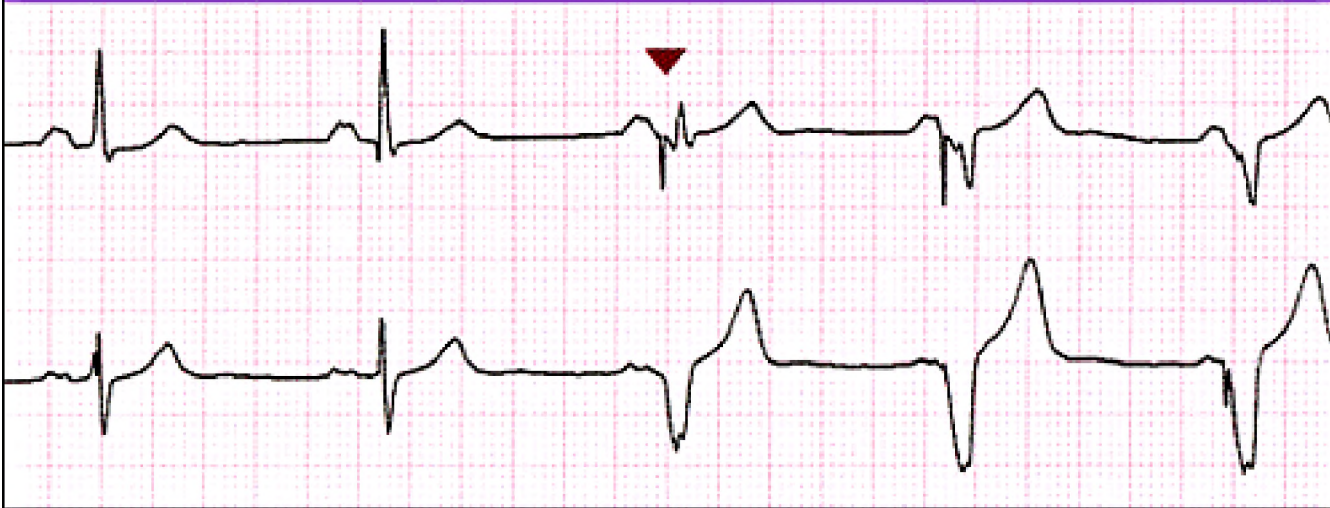
**The pacemaker does not generate a pacemaker spike  
when it is needed**

## Pacemaker Failure to Pace - Marquette-KH

*Marquette Electronics Copyright 1996*



## Paced Fusion Beat



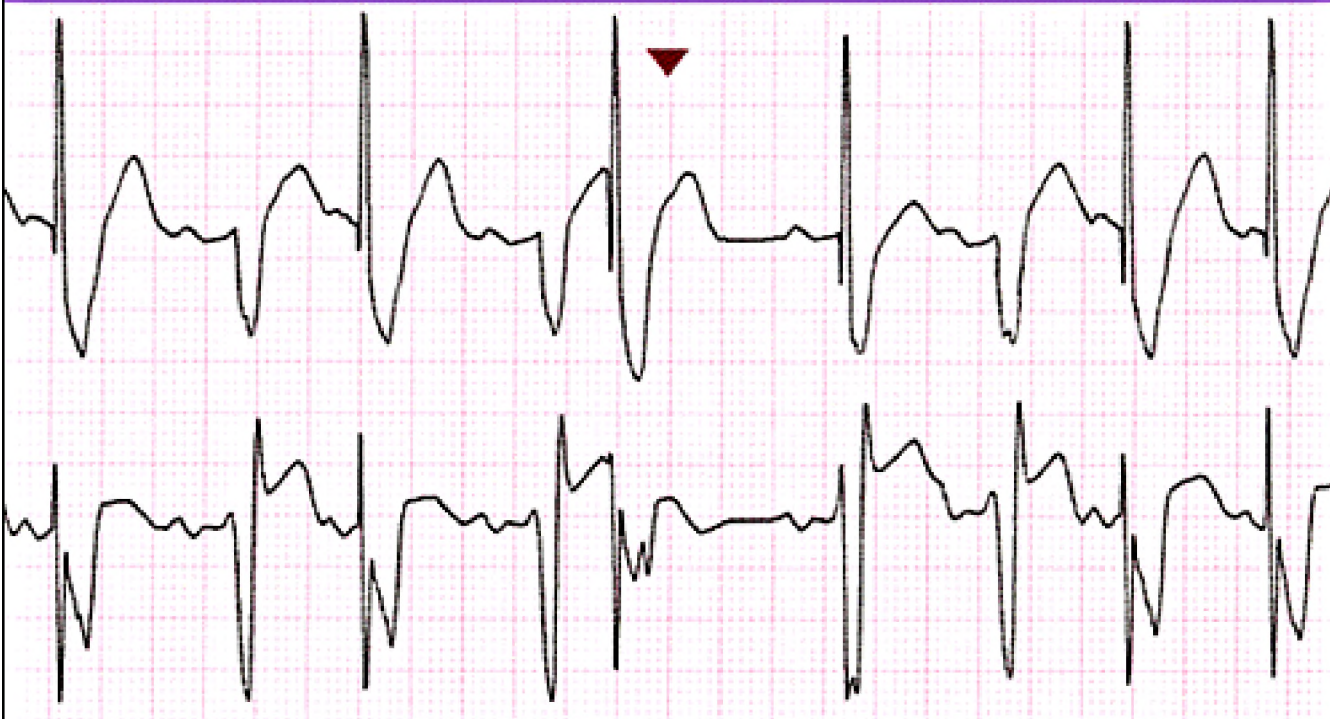
**The artificial pacemaker and the patient's own cardiac rhythm occurs simultaneously producing a combination of a paced beat and a normal beat.**

## Pacemaker Fusion Beat - Marquette-KH

*Marquette Electronics Copyright 1996*



## Failure to Sense



**The pacemaker does not recognize normal beats and generates an unnecessary pacemaker spike.**

## Pacemaker Failure To Sense - Marquette-KH

*Marquette Electronics Copyright 1996*

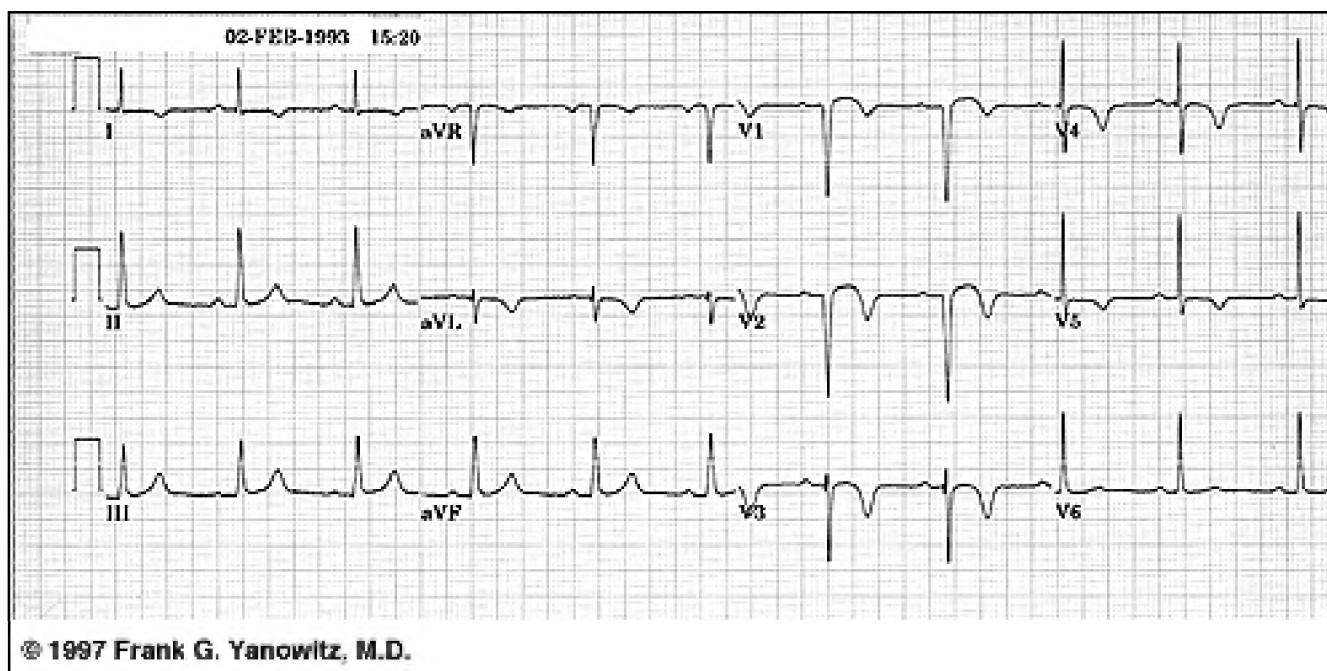
## Electronic Pacemaker Spikes



**Artificially induces electronic stimulus that paces the patient's rhythm causing a blip or spike on the ECG waveform**

## Electronic Ventricular Pacemaker Rhythm - Marquette-KH

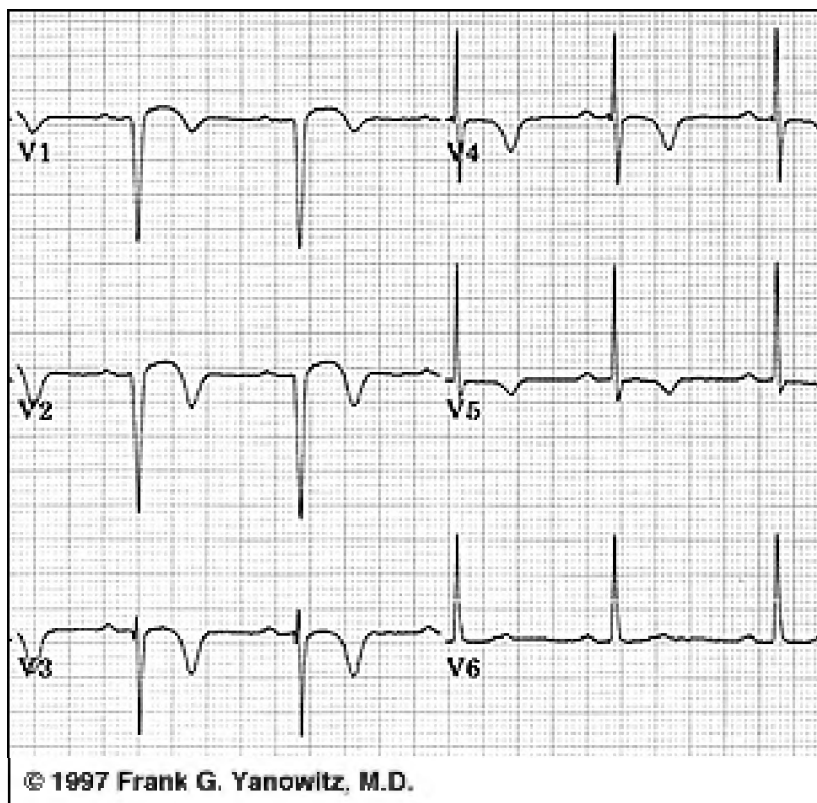
*Marquette Electronics Copyright 1996*



## Anteroseptal MI: Fully Evolved-KH

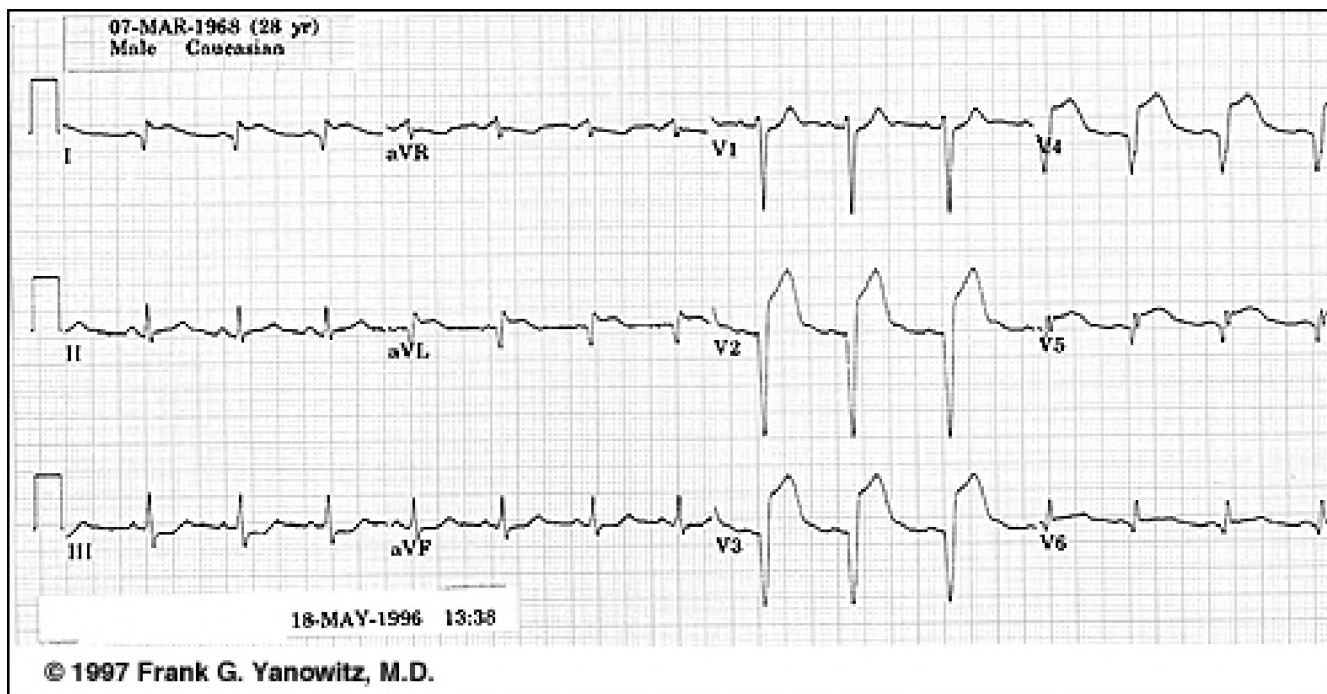
*Frank G. Yanowitz, M.D.*

The QS complexes, resolving ST segment elevation and T wave inversions in V1-2 are evidence for a fully evolved anteroseptal MI. The inverted T waves in V3-5, I, aVL are also probably related to the MI.



## Anteroseptal MI, Fully Evolved: Precordial Leads-KH

*Frank G. Yanowitz, M.D.*

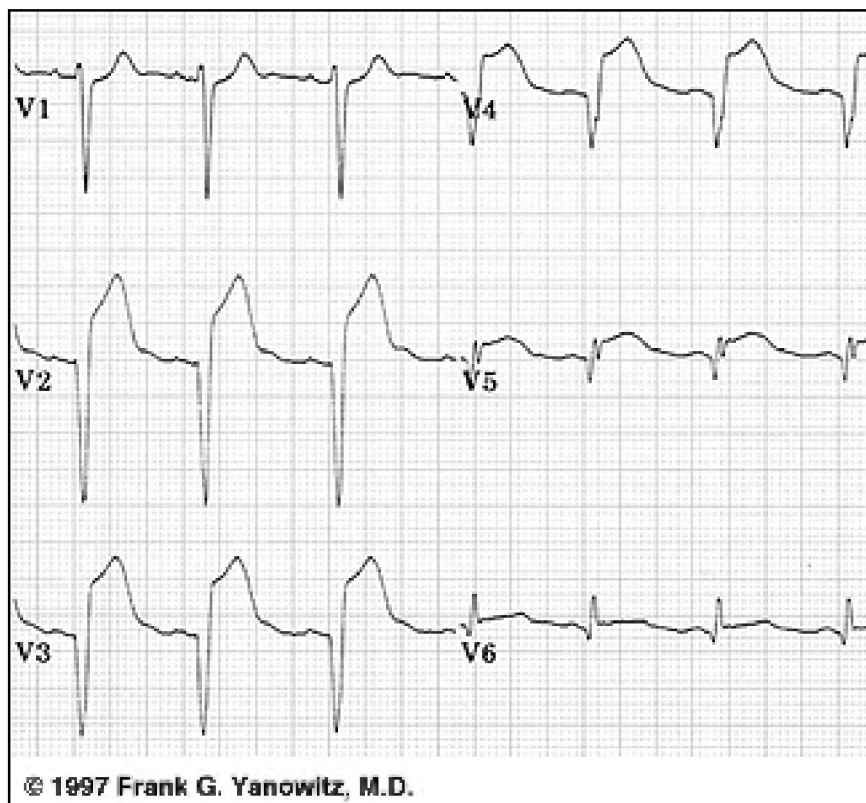


## Extensive Anterior/Anterolateral MI: Recent-KH

*Frank G. Yanowitz, M.D.*

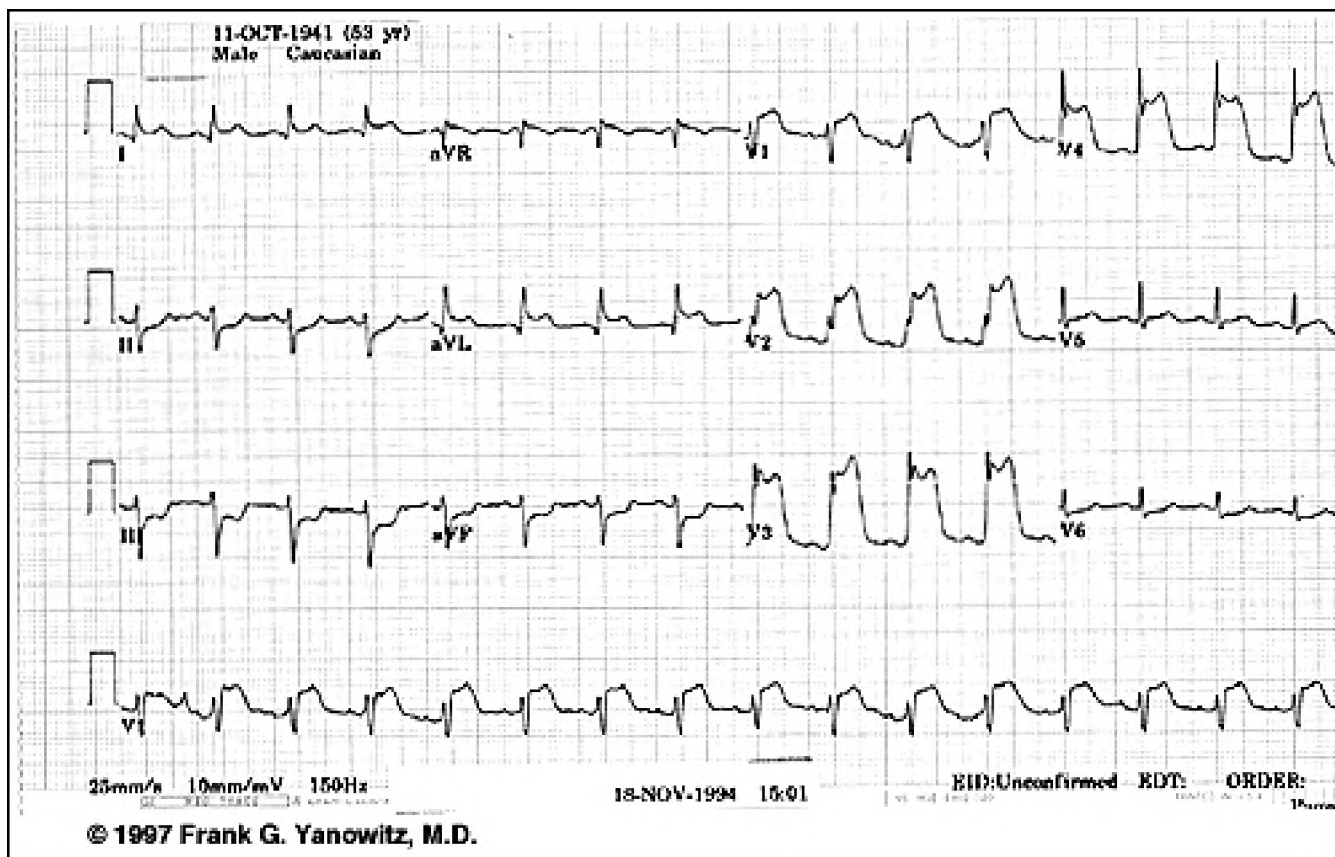
Significant pathologic Q-waves (V2-6, I, aVL) plus marked ST segment elevation are evidence for this large anterior/anterolateral MI. The exact age of the infarction cannot be determined without clinical correlation and previous ECGs, but this is likely a recent MI.





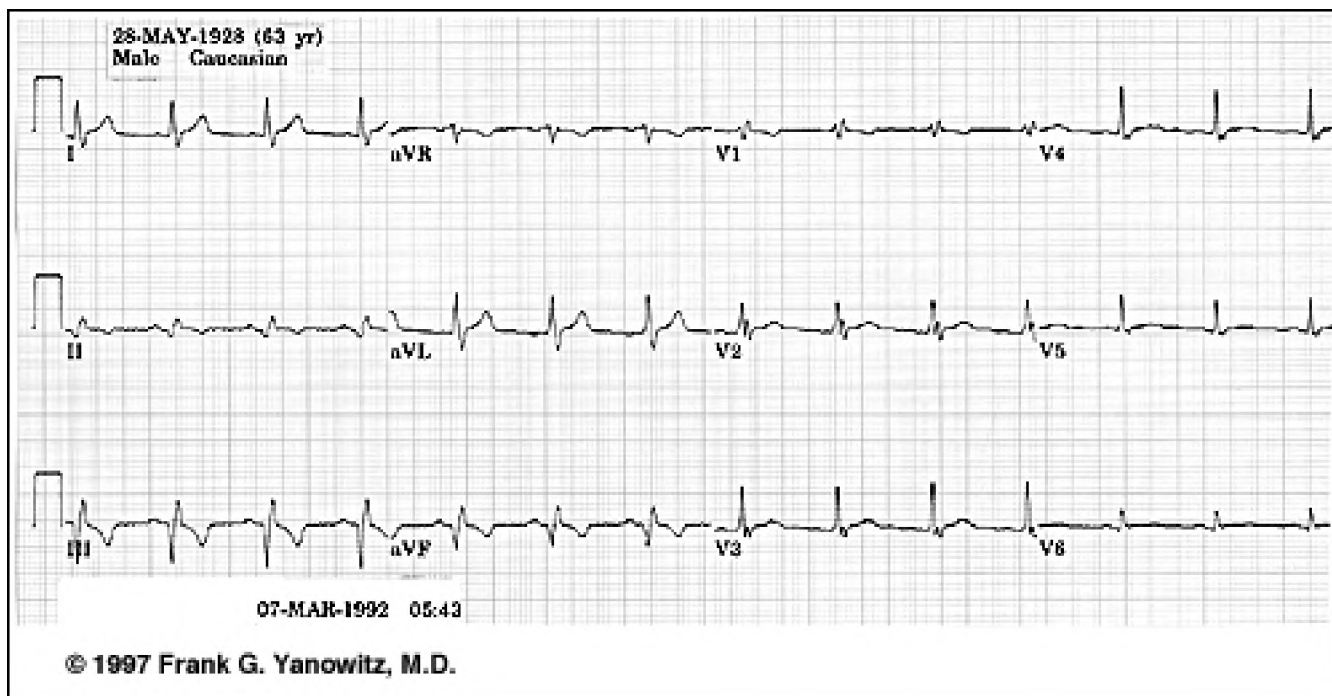
## Extensive Anterior/Anterolateral MI: Precordial Leads-KH

*Frank G. Yanowitz, M.D.*



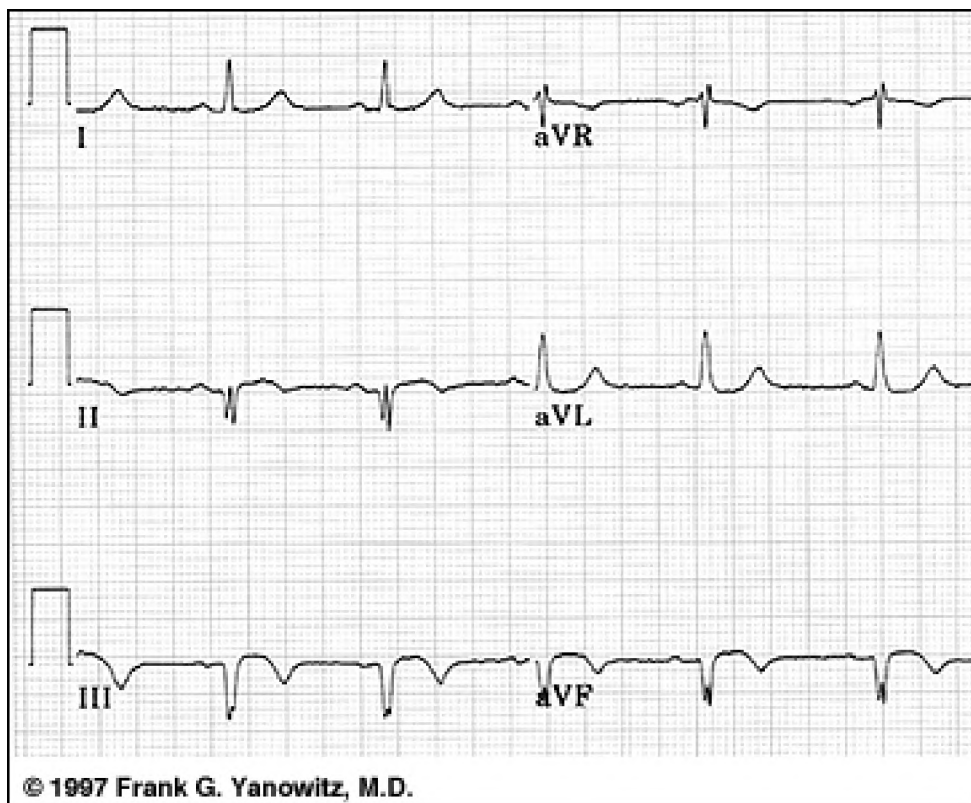
## Acute Anterior MI-KH

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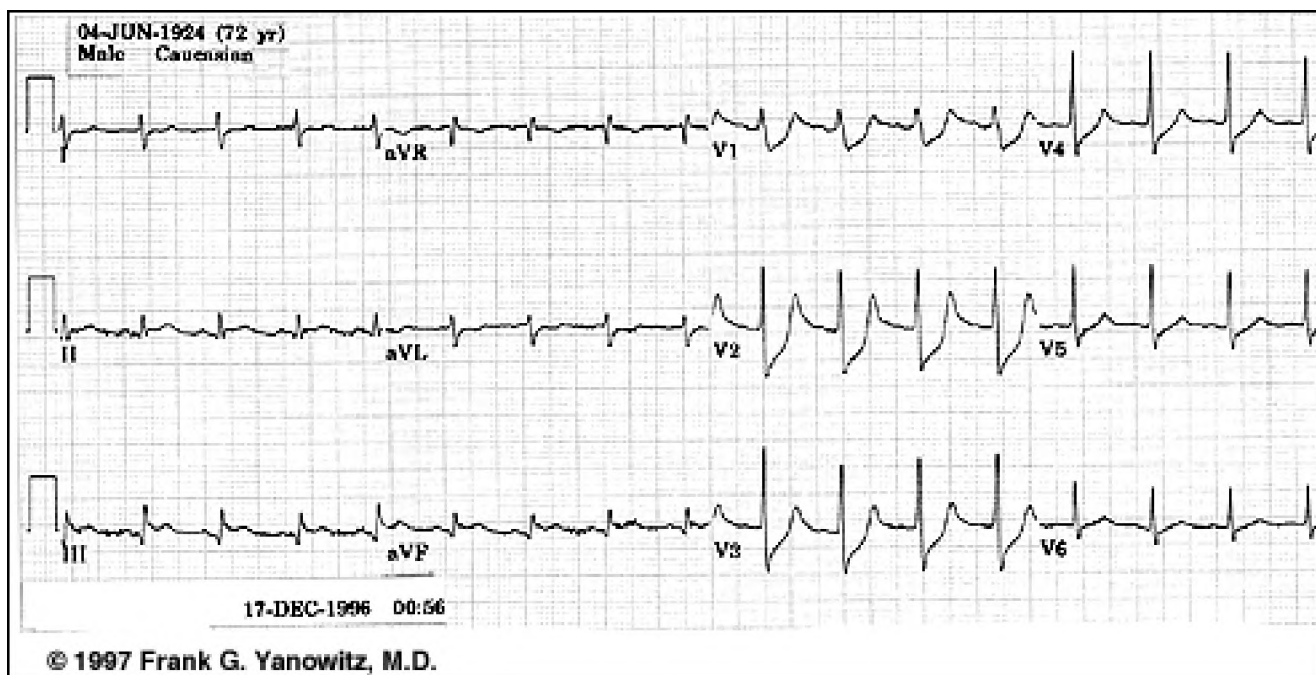
## Infero-posterior MI-KH

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## Fully Evolved Inferior MI: Frontal Plane-KH

*Frank G. Yanowitz, M.D.*



## Acute Inferoposterior MI-KH

*Frank G. Yanowitz, M.D., copyright 1997*

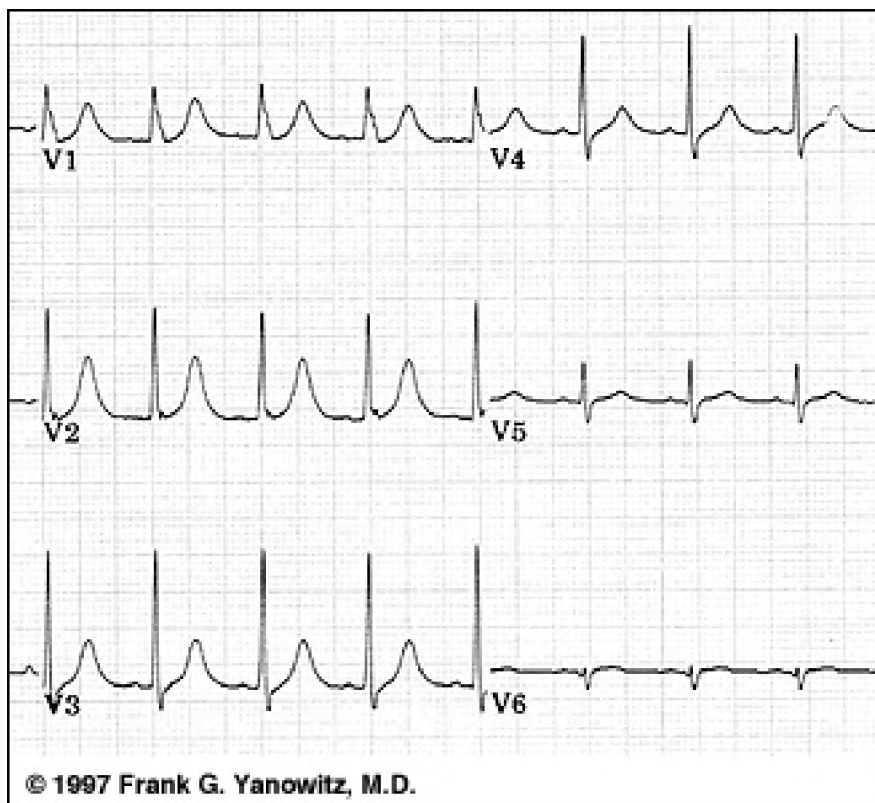




## Postero-lateral MI: Fully Evolved-KH

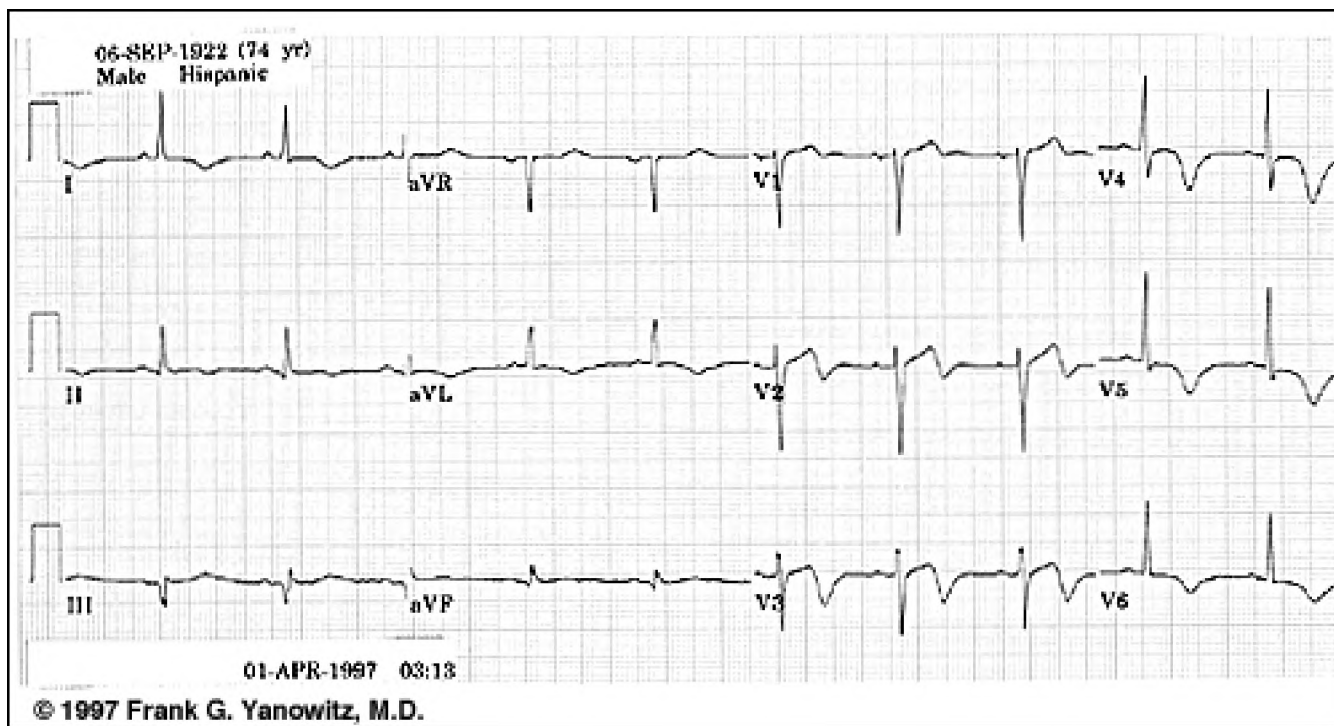
*Frank G. Yanowitz, M.D.*

The "true" posterior MI is recognized by pathologic R waves in leads V1-2. These are the posterior equivalent of pathologic Q waves (seen from the perspective of the anterior leads). Tall T waves in these same leads are the posterior equivalent of inverted T waves in this fully evolved MI. The loss of forces in V6, I, aVL suggest a lateral wall extension of this MI.



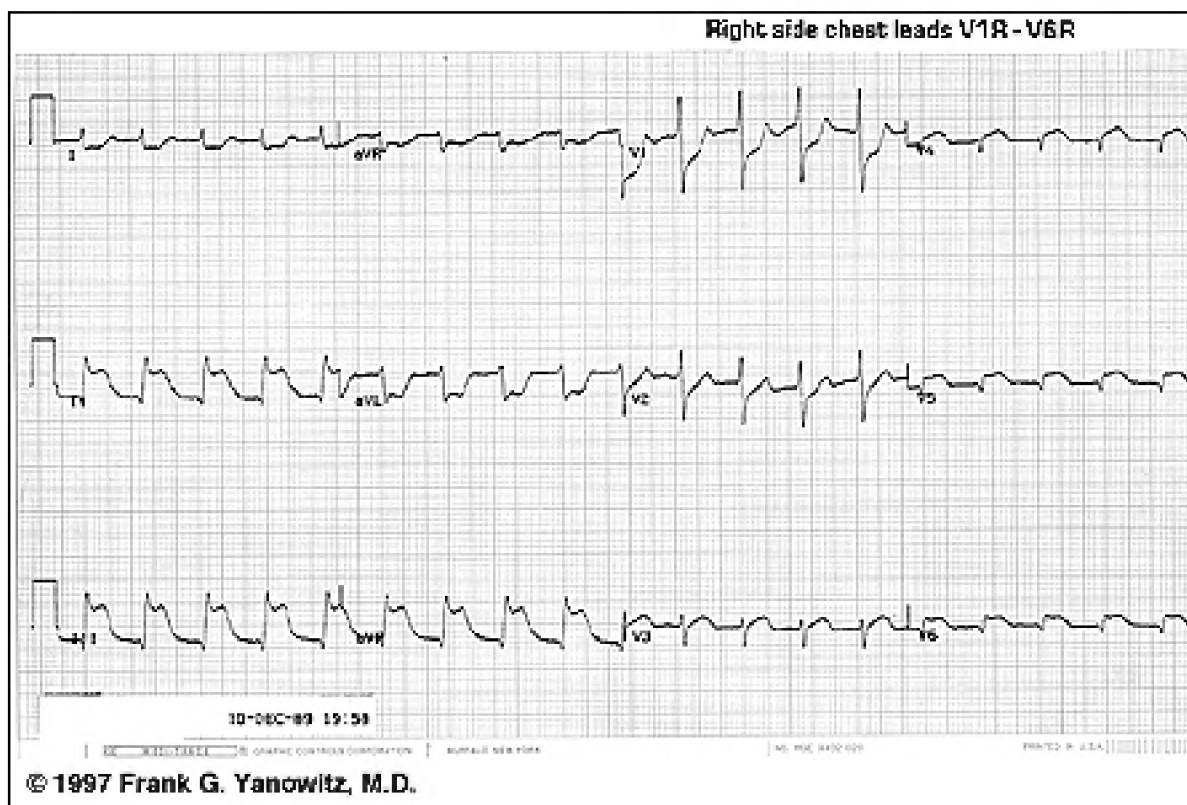
## Postero-lateral MI: Precordial Leads-KH

*Frank G. Yanowitz, M.D.*



## Diffuse Anterolateral T Wave Abnormalities-KH

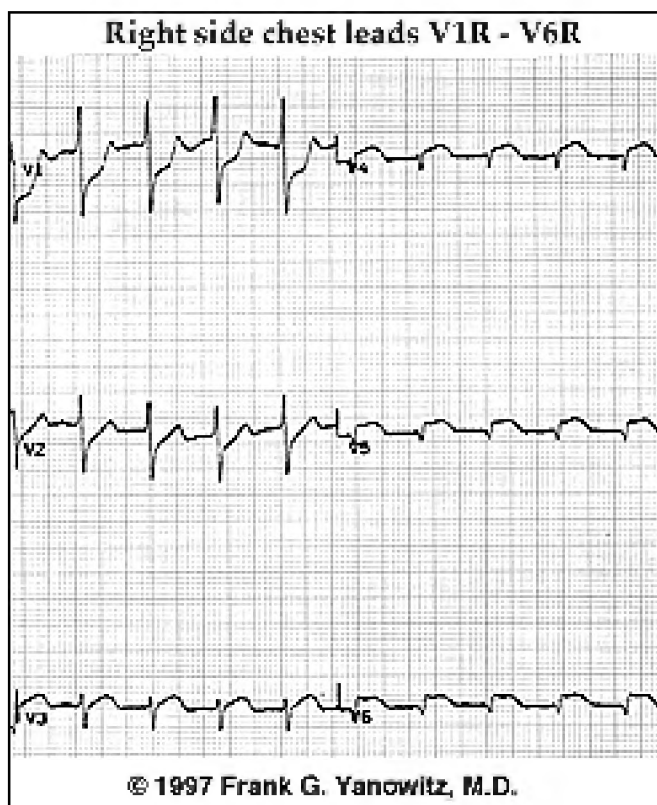
*Frank G. Yanowitz, M.D., copyright 1997*



# Acute Inferoposterior MI with Right Ventricular MI

*Frank G. Yanowitz, M.D.*

Hyperacute ST segment elevation is seen in leads II, III, aVF (inferior location) and ST depression is seen in leads V1-2 (an expression of posterior wall injury). Right precordial leads V1R - V6R illustrate right ventricular infarction when ST segment elevation occurs in V3R or adjacent right precordial leads. Reciprocal ST segment depression is seen in leads I and aVL.

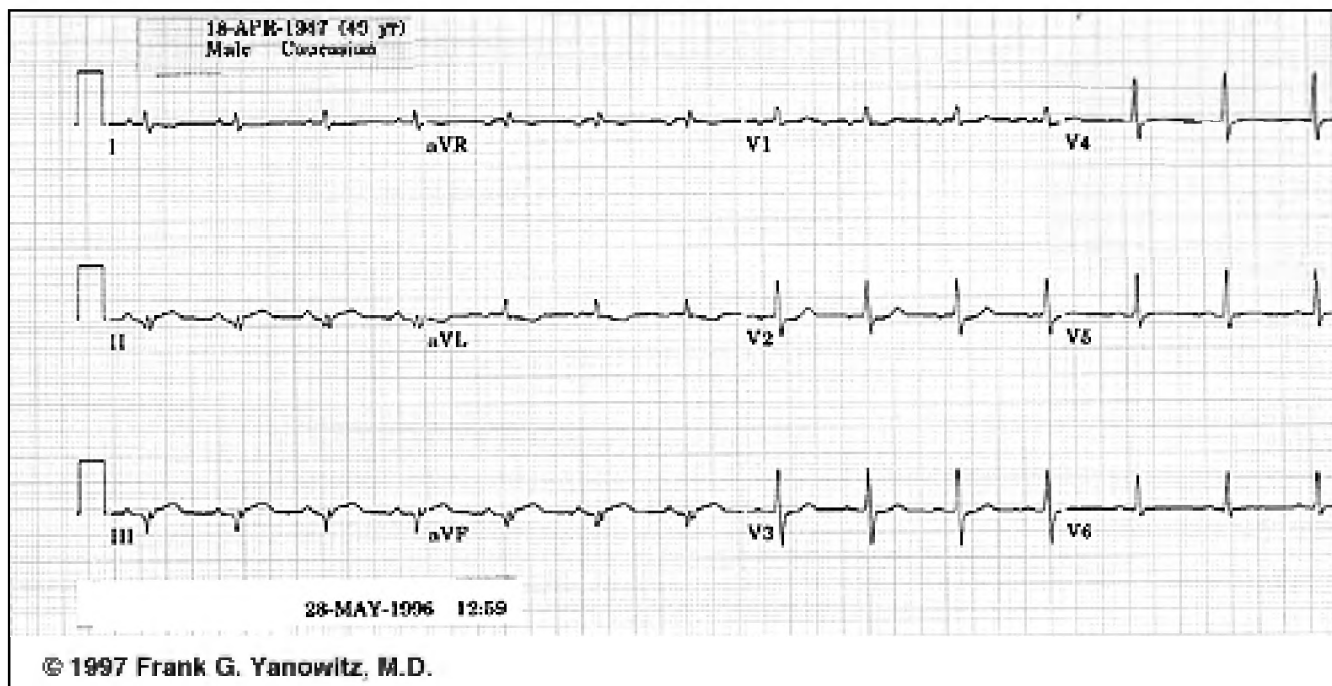


## True Posterior MI and Right Ventricular MI

*Frank G. Yanowitz, M.D.*

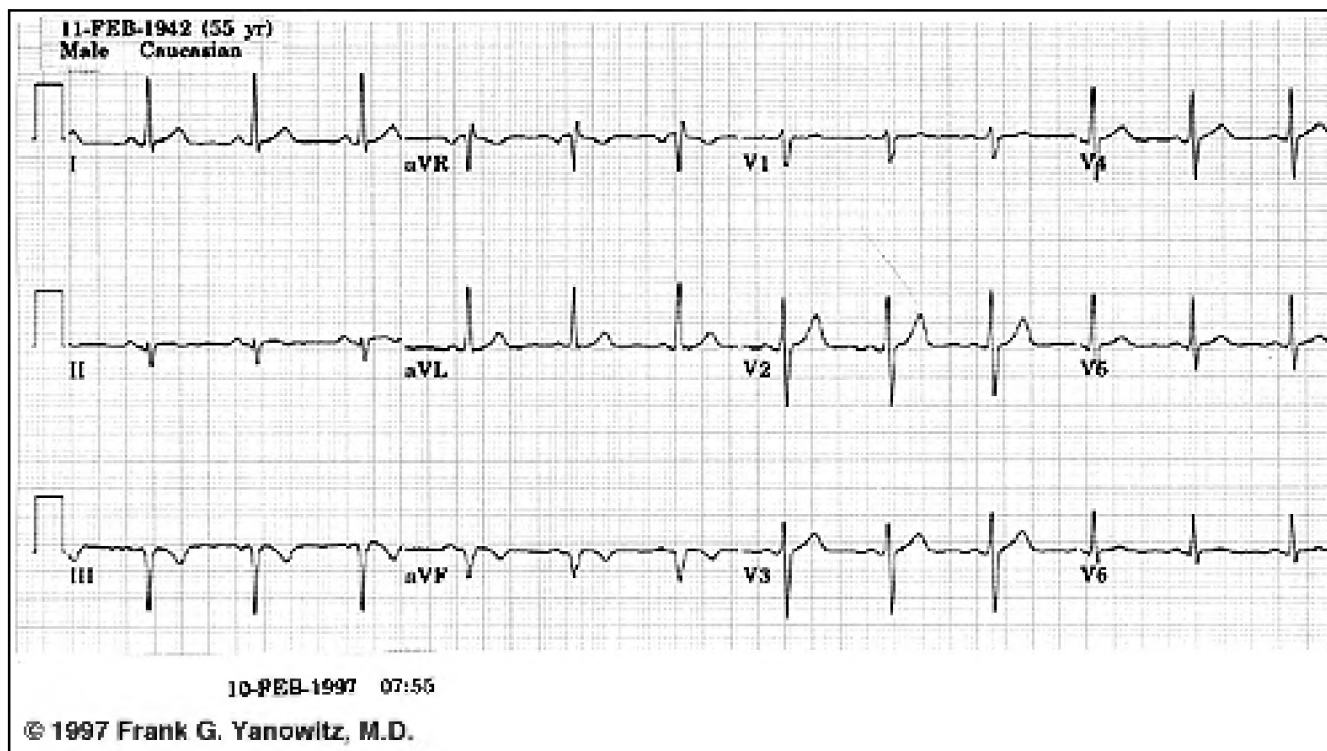
Right sided chest leads, V1R - V6R are shown. The true posterior MI is evidenced by the marked ST segment elevation in V1R (actual V2) and V2R (actual V1). The RV MI is evidenced by the ST elevation in V3R to V6R.





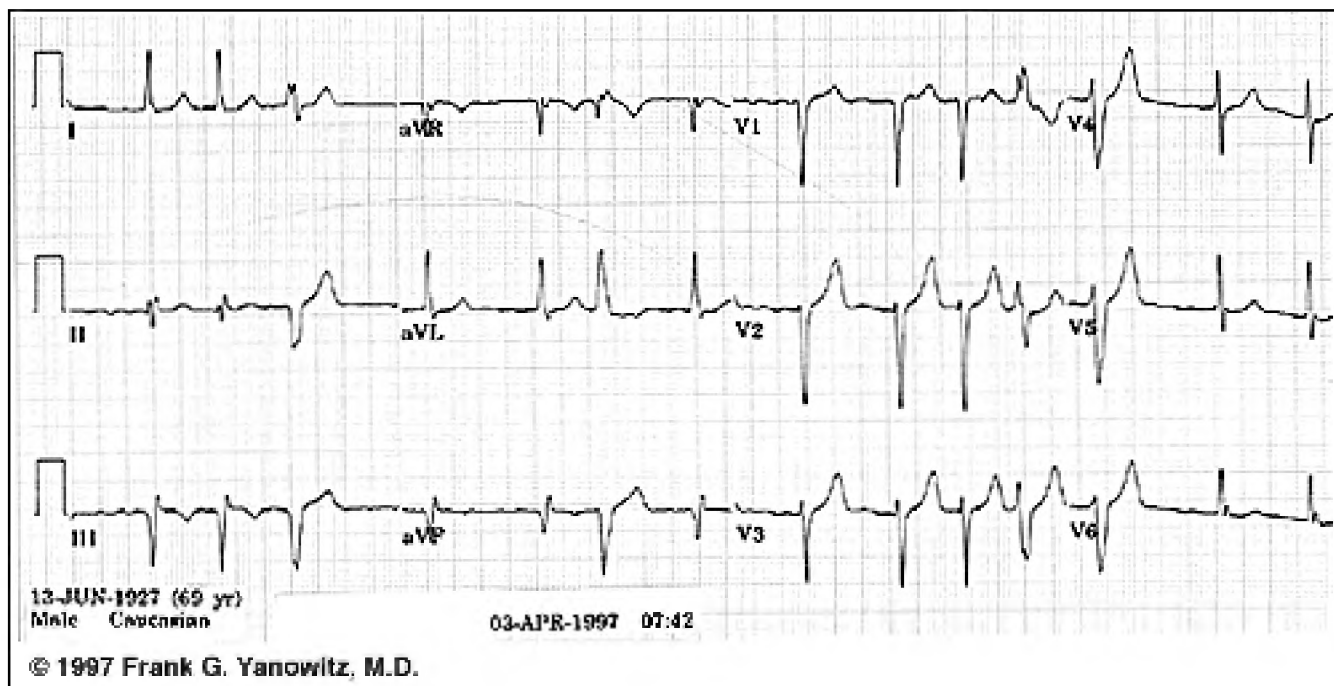
## Old Infero-posterior MI-KH

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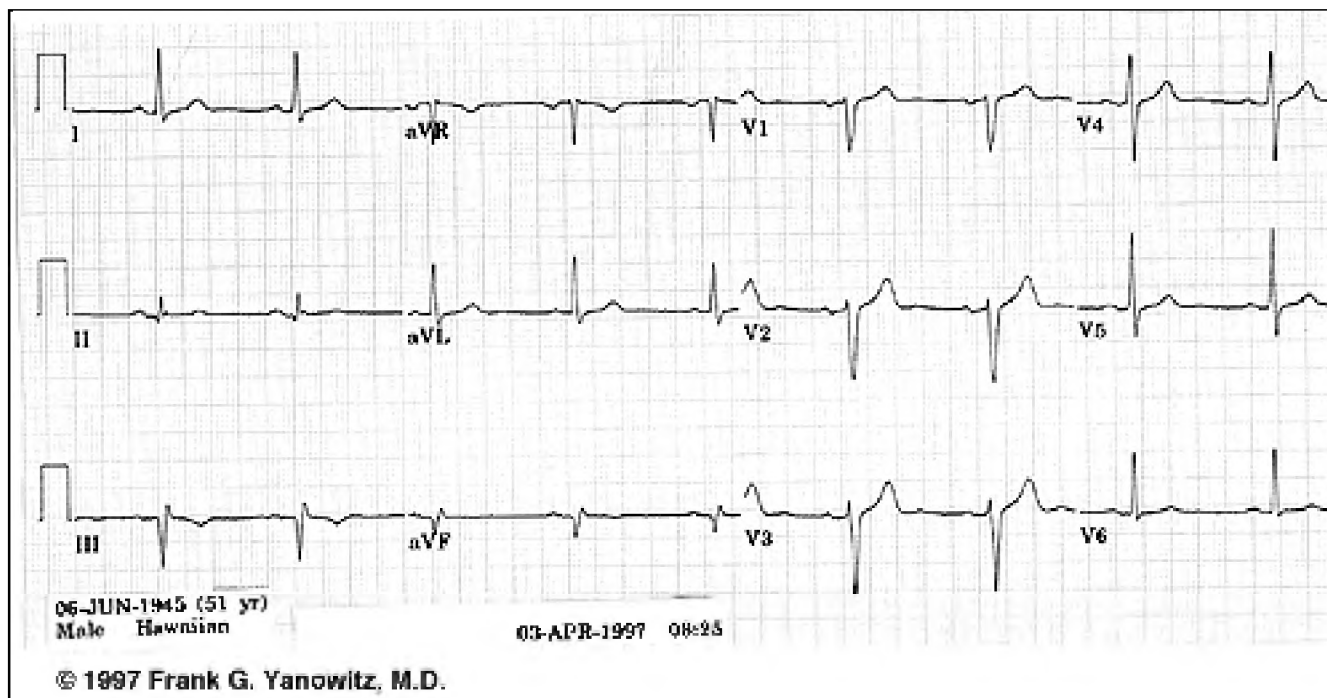
## Old Inferior MI-KH

*Frank G. Yanowitz, M.D., copyright 1997*



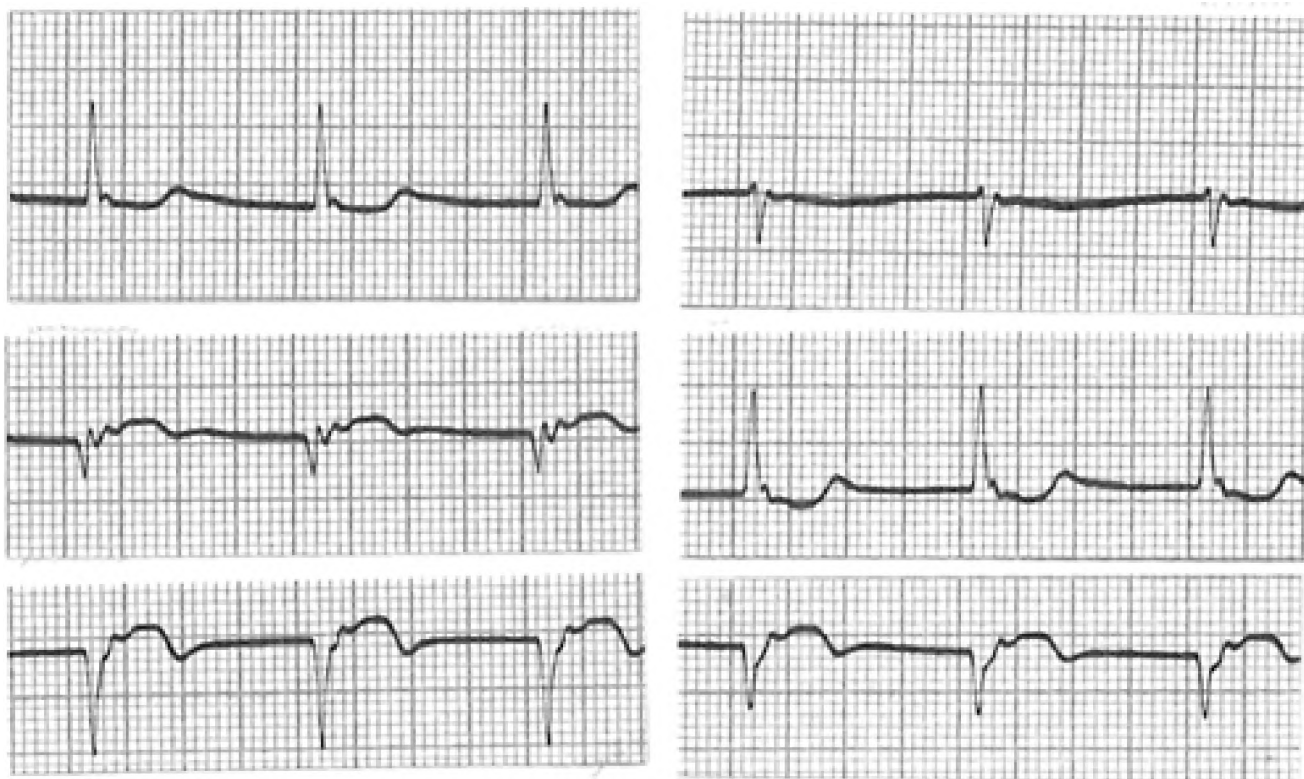
## Old Inferior MI, PVCs, and Atrial Fibrillation-KH

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## Old Inferior MI-KH

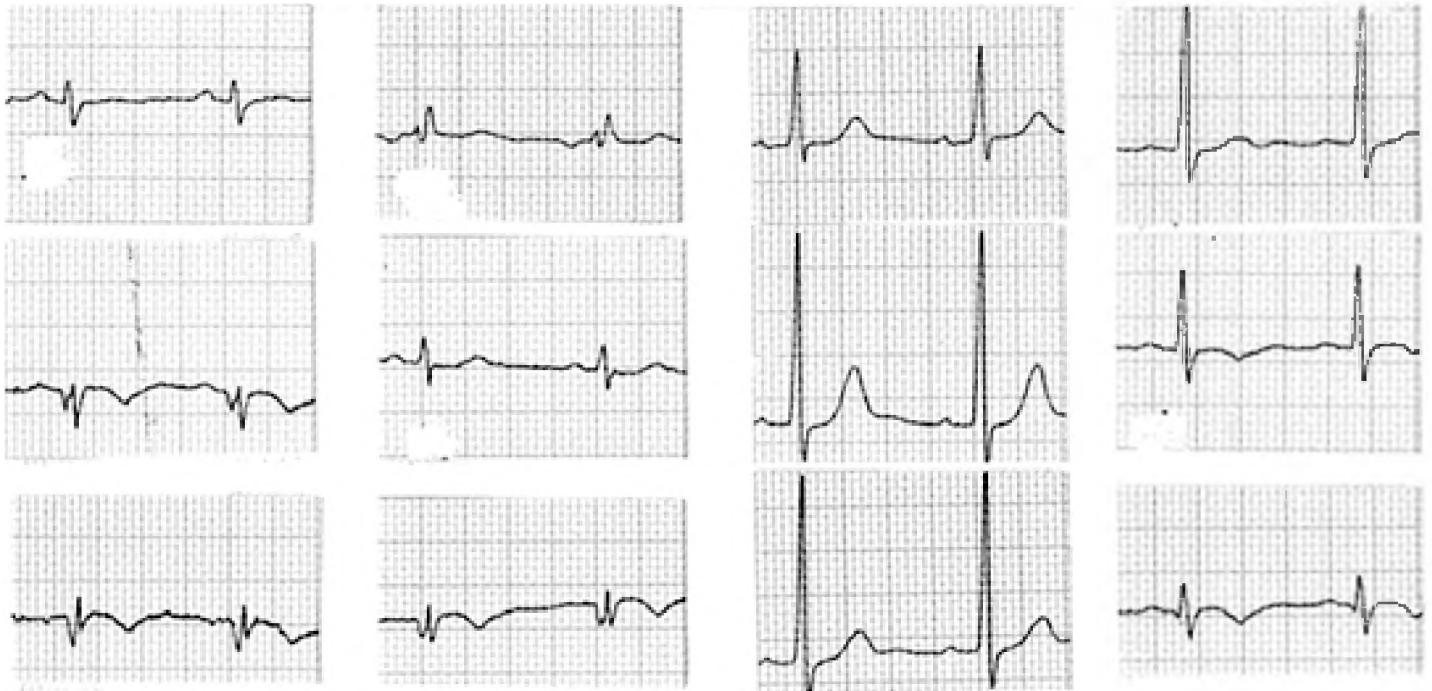
*Frank G. Yanowitz, M.D., copyright 1997*



## Frontal Plane: Accelerated Junctional Rhythm and Inferior MI-KH

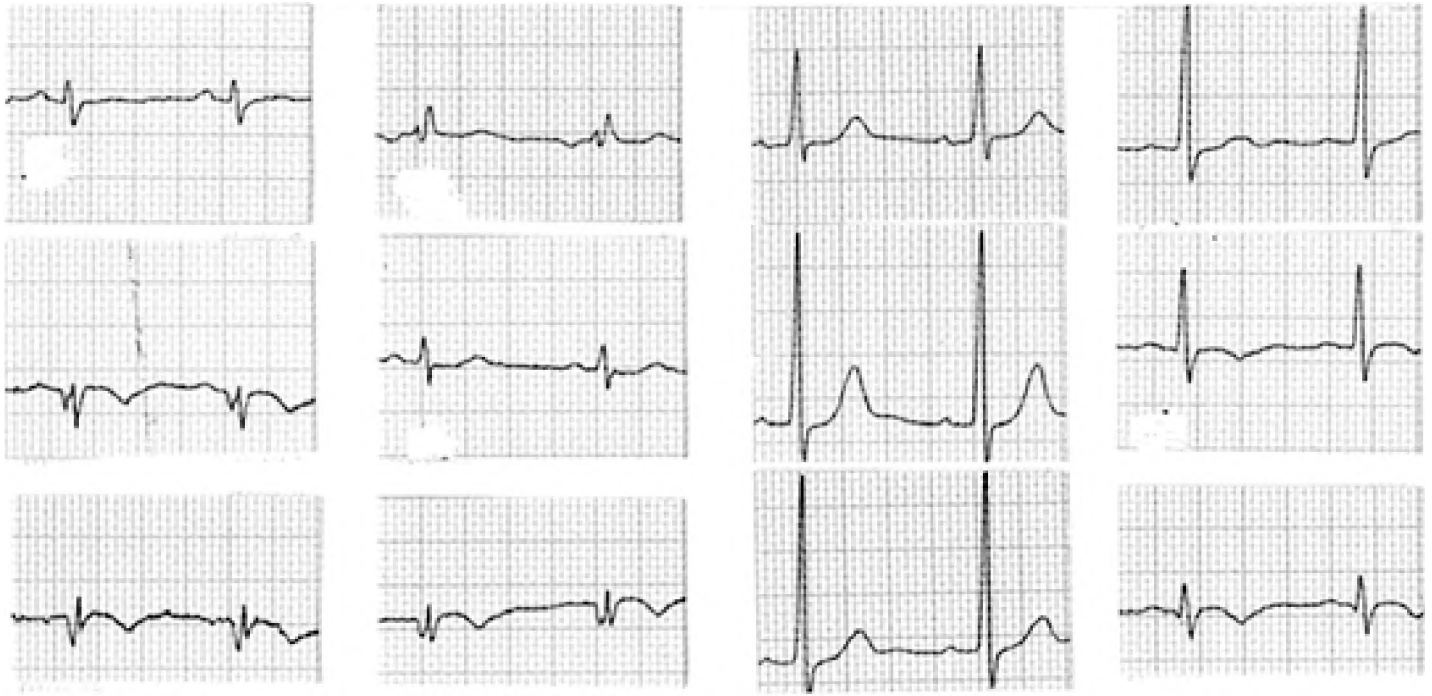
*Frank Yanowitz Copyright 1996*





## Inferoposterior MI-KH

*Frank Yanowitz Copyright 1996*



## Inferoposterior MI-KH

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### Lead II



### Lead V<sub>1</sub>



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## Left Atrial Abnormality & 1st Degree AV Block: Leads II and V<sub>1</sub>-KH

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## Lead II



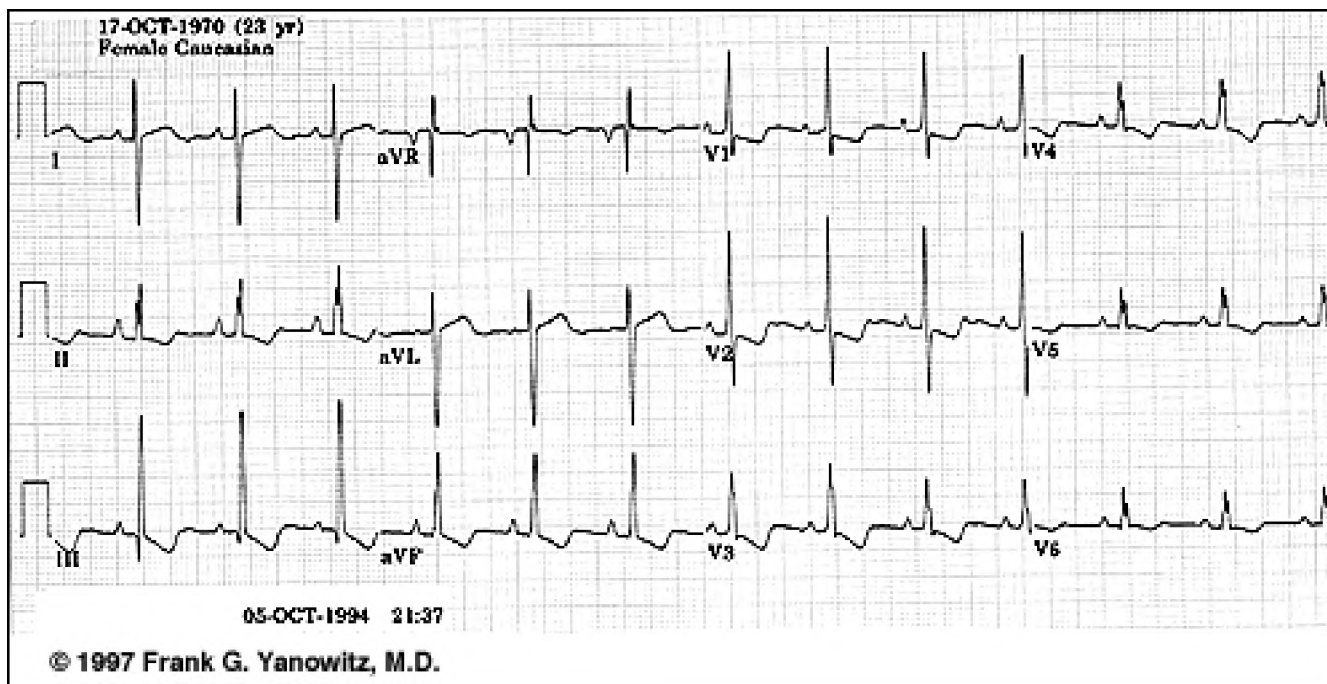
## Lead V<sub>1</sub>



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# Left Atrial Enlargement: Leads II and V1-KH

*Frank G. Yanowitz, M.D.*



## Right Ventricular Hypertrophy (RVH) & Right Atrial Enlargement (RAE)-KH

*Frank G. Yanowitz, M.D.*

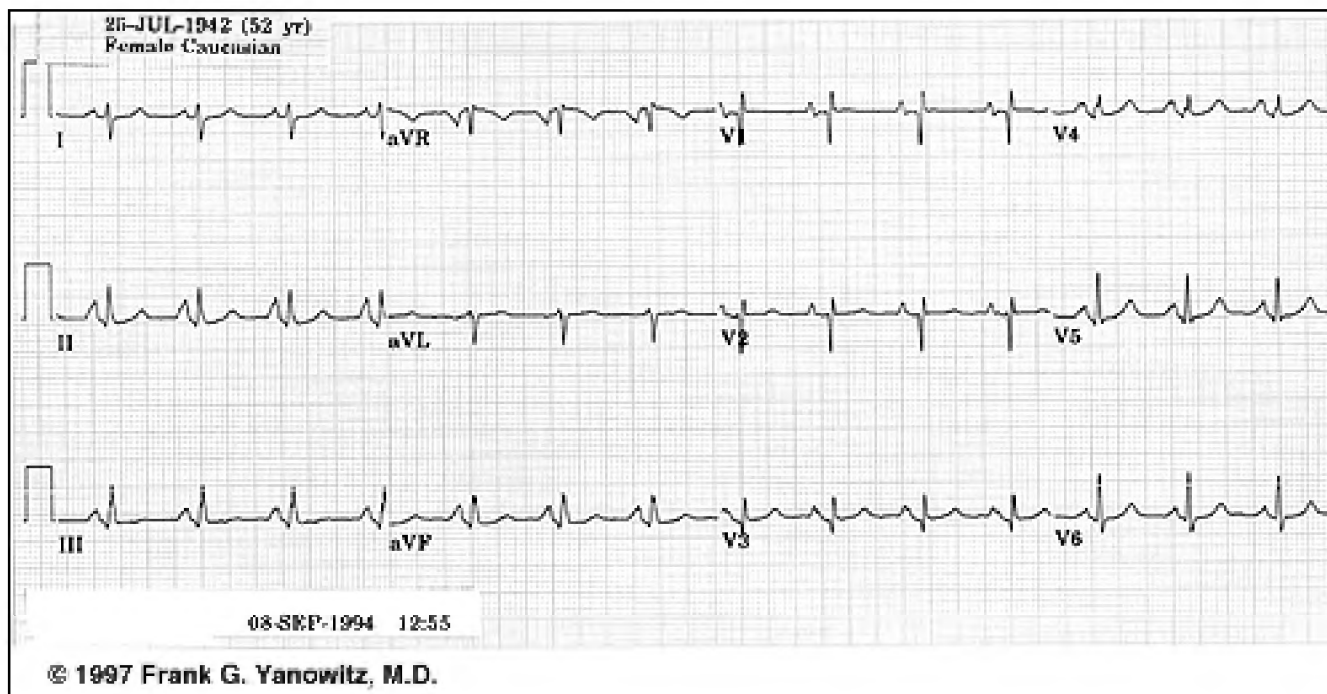
In this case of severe pulmonary hypertension, RVH is recognized by the prominent anterior forces (tall R waves in V1-2), right axis deviation (+110 degrees), and "P pulmonale" (i.e., right atrial enlargement). RAE is best seen in the frontal plane leads; the P waves in lead II are >2.5mm in amplitude.





## Right Axis Deviation & RAE (P Pulmonale): Leads I, II, III-KH

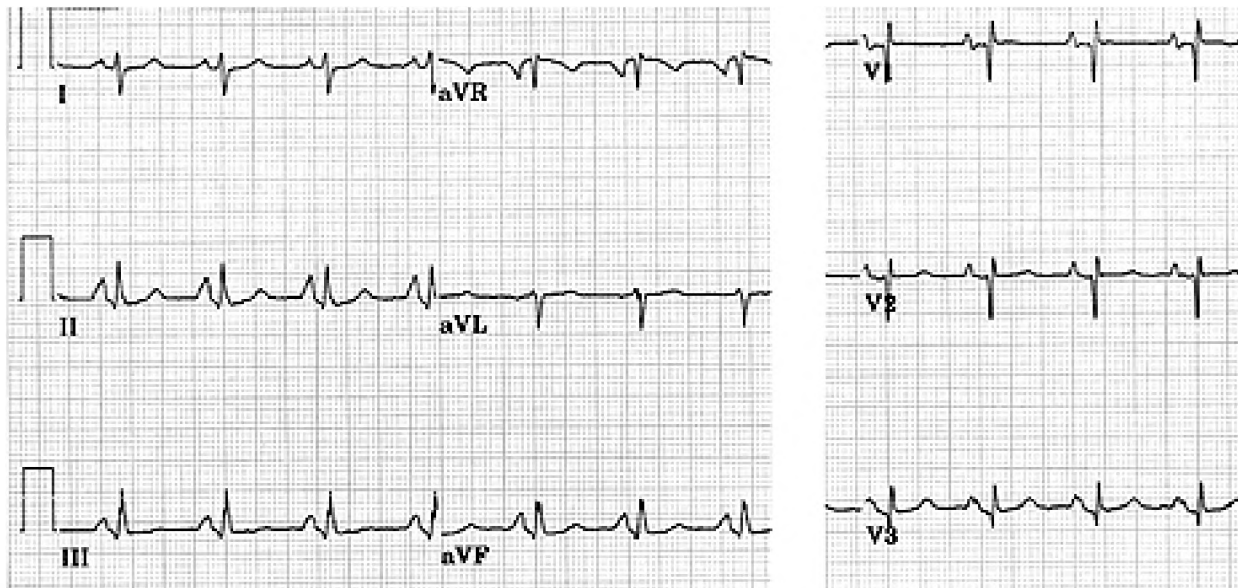
*Frank G. Yanowitz, M.D.*



## Right Atrial Enlargement (RAE) & Right Ventricular Hypertrophy (RVH)-KH

*Frank G. Yanowitz, M.D.*

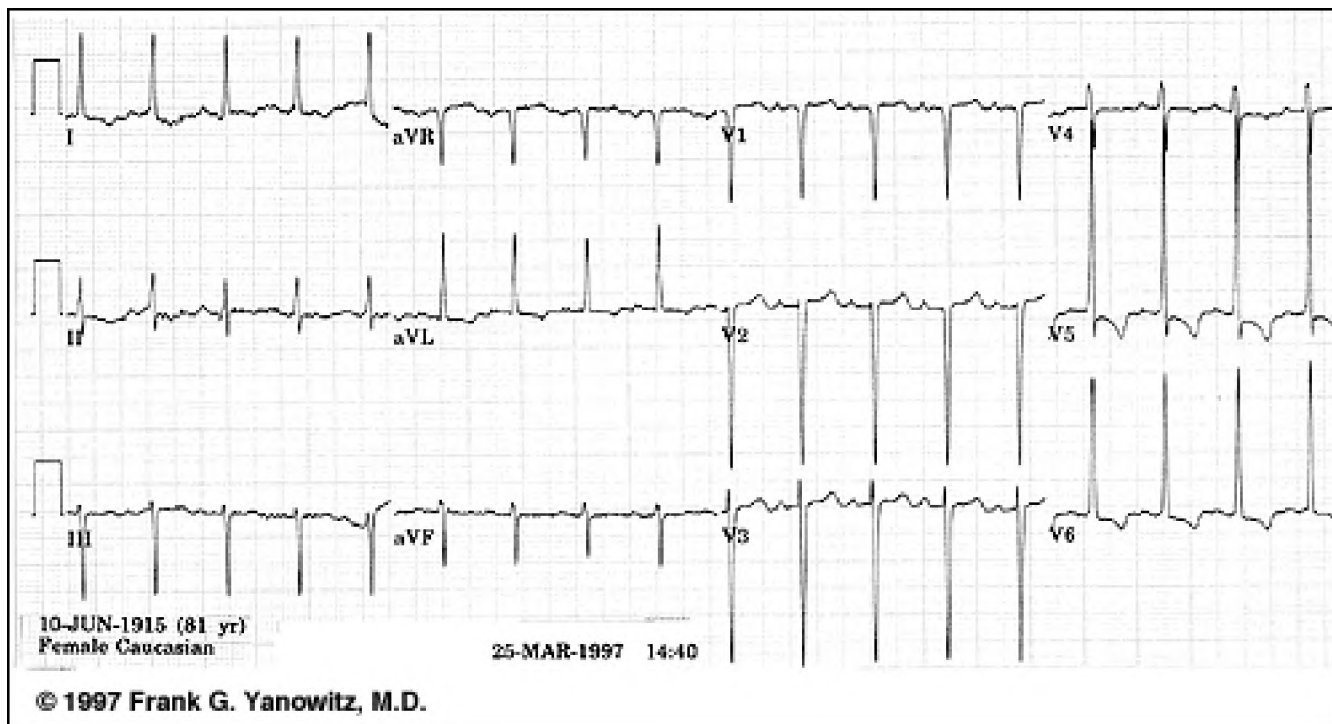
RAE is recognized by the tall ( $>2.5\text{mm}$ ) P waves in leads II, III, aVF. RVH is likely because of right axis deviation ( $+100$  degrees) and the Qr (or rSR') complexes in V1-2.



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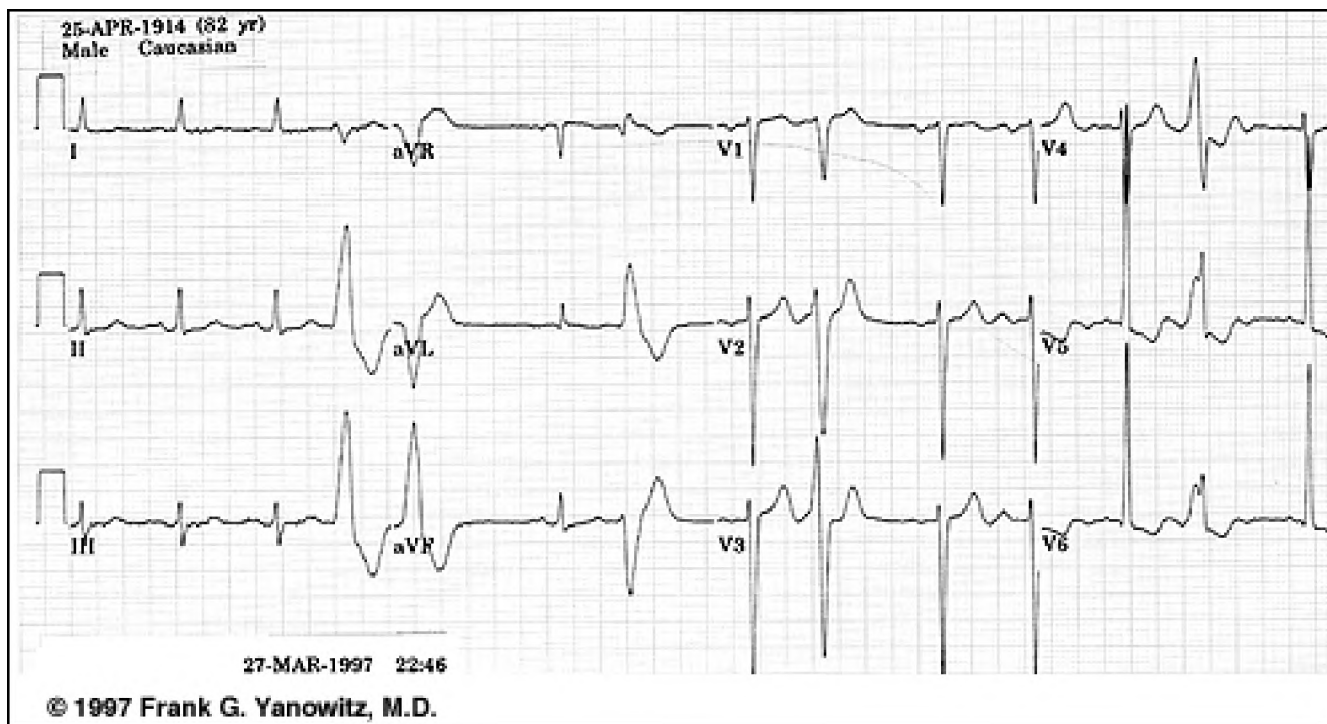
## RAE & RVH-KH

*Frank G. Yanowitz, M.D.*



## LVH with "Strain"-KH

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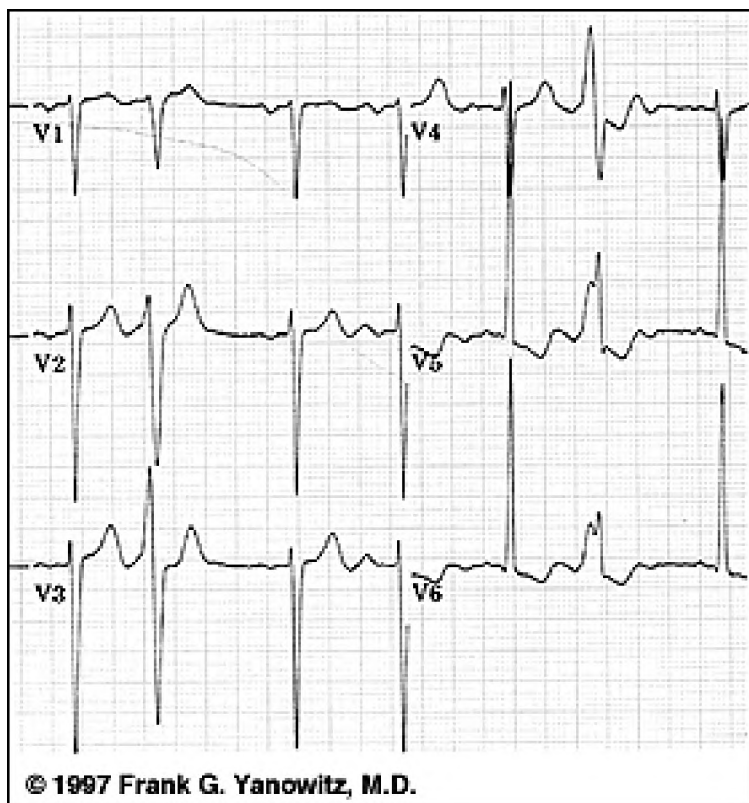


## LVH and Many PVCs-KH

*Frank G. Yanowitz, M.D.*

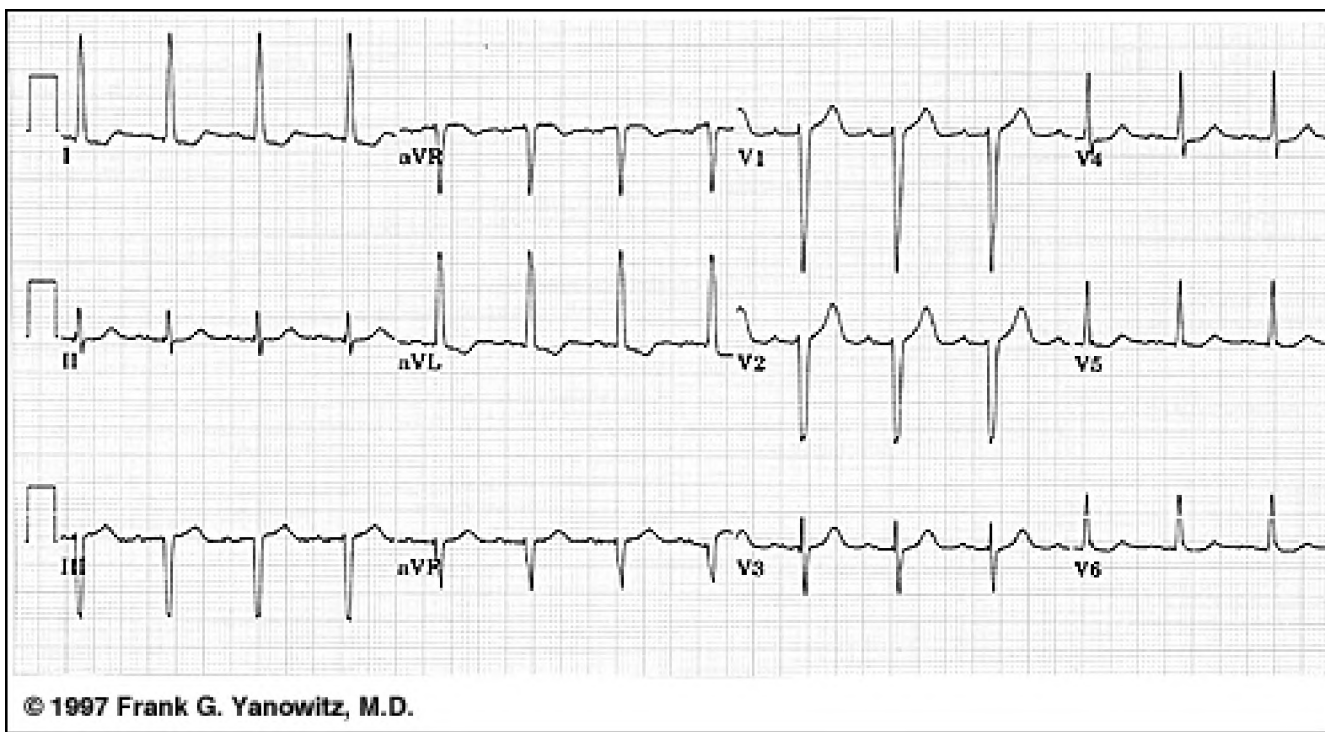
The combination of voltage criteria ( $SV_2 + RV_6 > 35\text{mm}$ ) and ST-T abnormalities in V5-6 are definitive for LVH. There may also be LAE as evidenced by the prominent negative P terminal force in lead V1. Isolated PVCs and a PVC couplet are also present.





## LVH & PVCs: Precordial Leads-KH

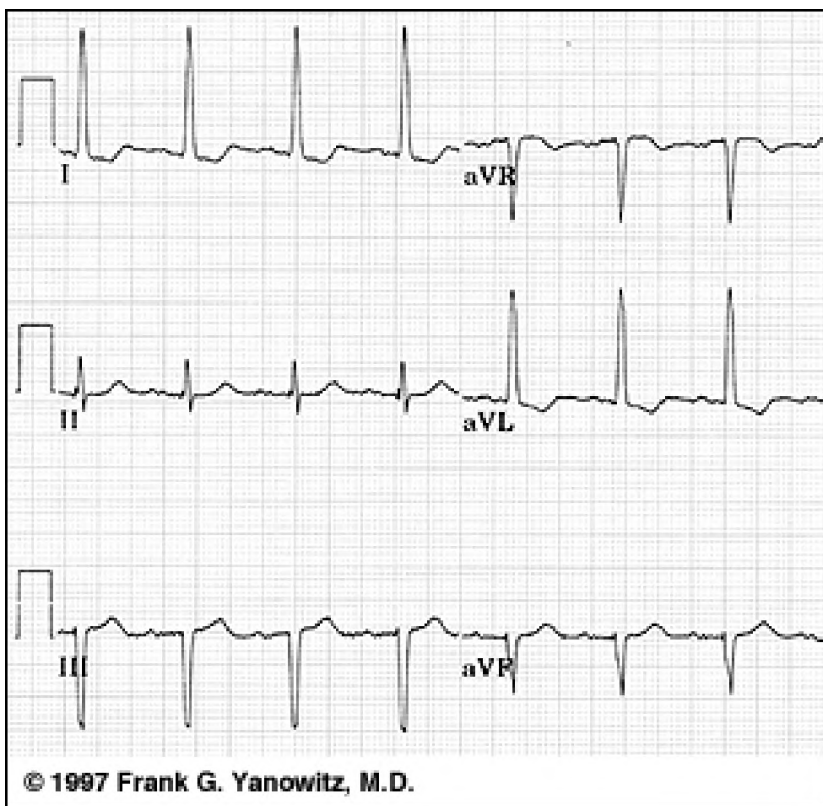
*Frank G. Yanowitz, M.D.*



## LVH: Limb Lead Criteria-KH

*Frank G. Yanowitz, M.D.*

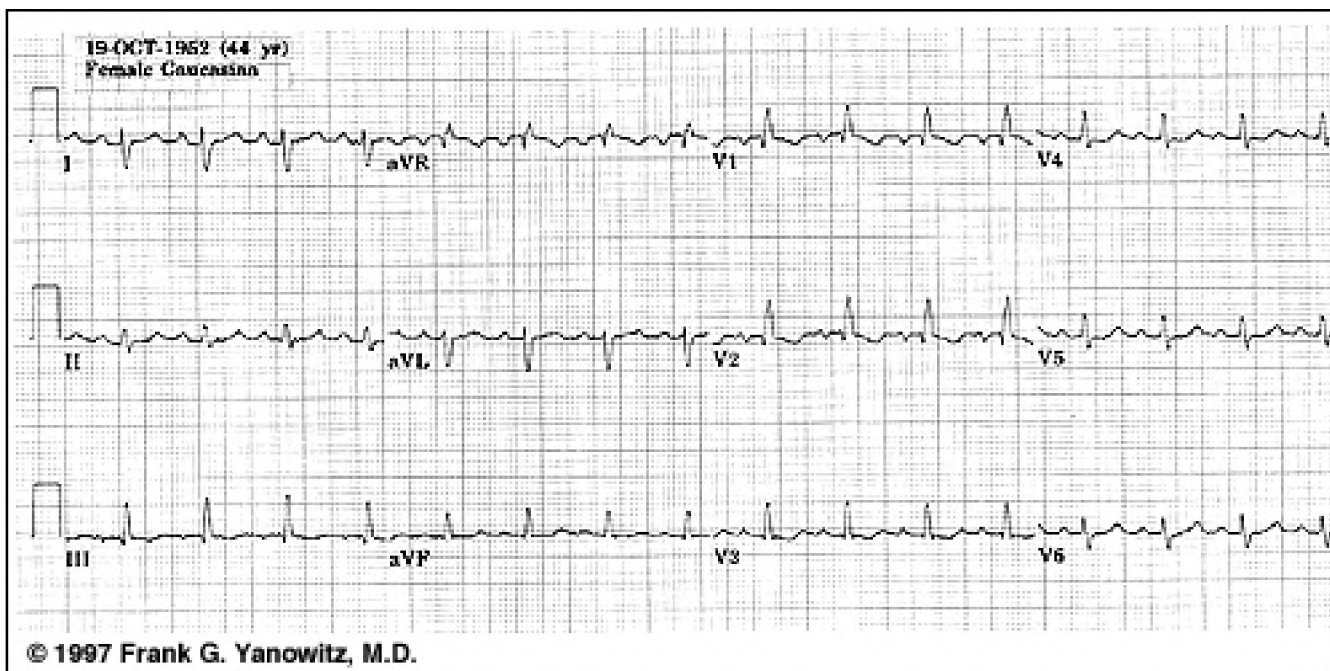
In this example of LVH, the precordial leads don't meet the usual voltage criteria or exhibit significant ST segment abnormalities. The frontal plane leads, however, show voltage criteria for LVH and significant ST segment depression in leads with tall R waves. The voltage criteria include 1) R in aVL >11 mm; 2) R in I + S in III >25mm; and 3)  $(RI+SIII) - (RIII+SI) >17\text{mm}$  (Lewis Index).



## LVH: Limb Lead Criteria-KH

*Frank G. Yanowitz, M.D.*

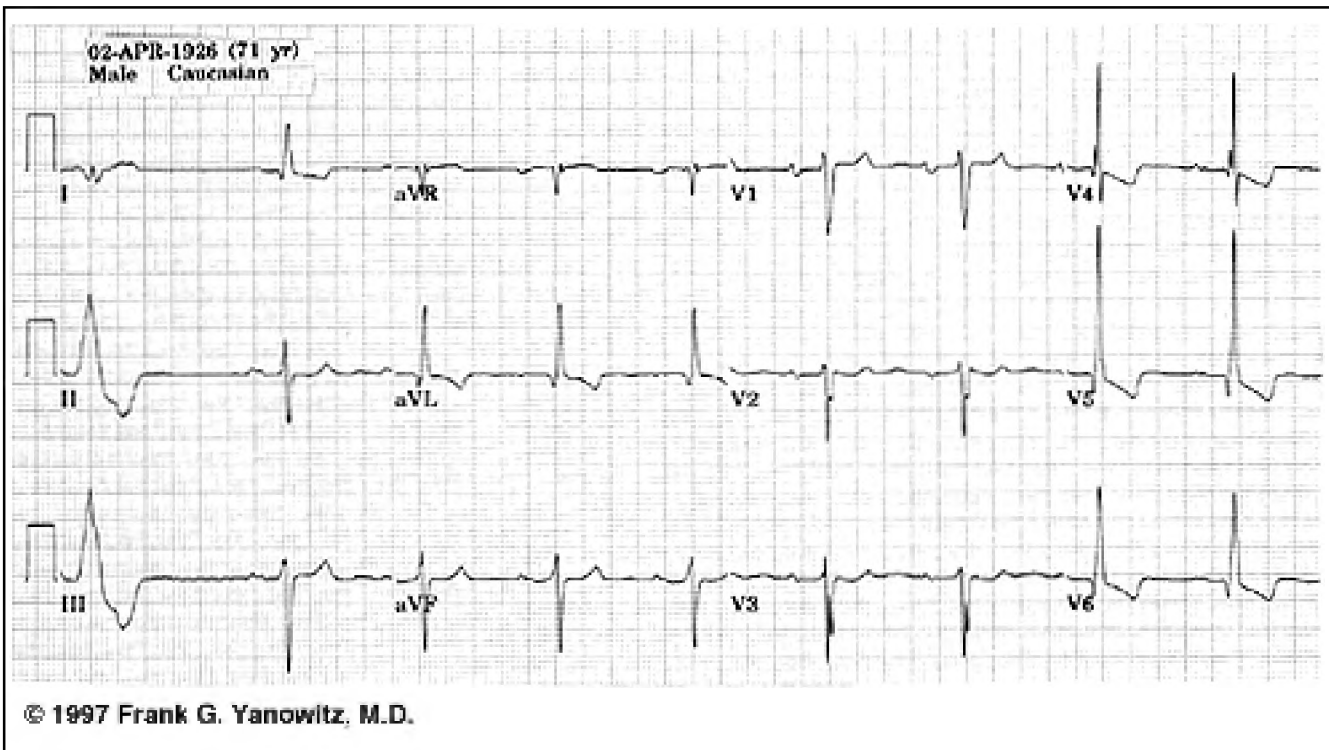
In this example of LVH, the precordial leads don't meet the usual voltage criteria or exhibit significant ST segment abnormalities. The frontal plane leads, however, show voltage criteria for LVH and significant ST segment depression in leads with tall R waves. The voltage criteria include 1) R in aVL >11 mm; 2) R in I + S in III >25mm; and 3) (RI+SIII) - (RIII+SI) >17mm (Lewis Index).



## RVH with Right Axis Deviation

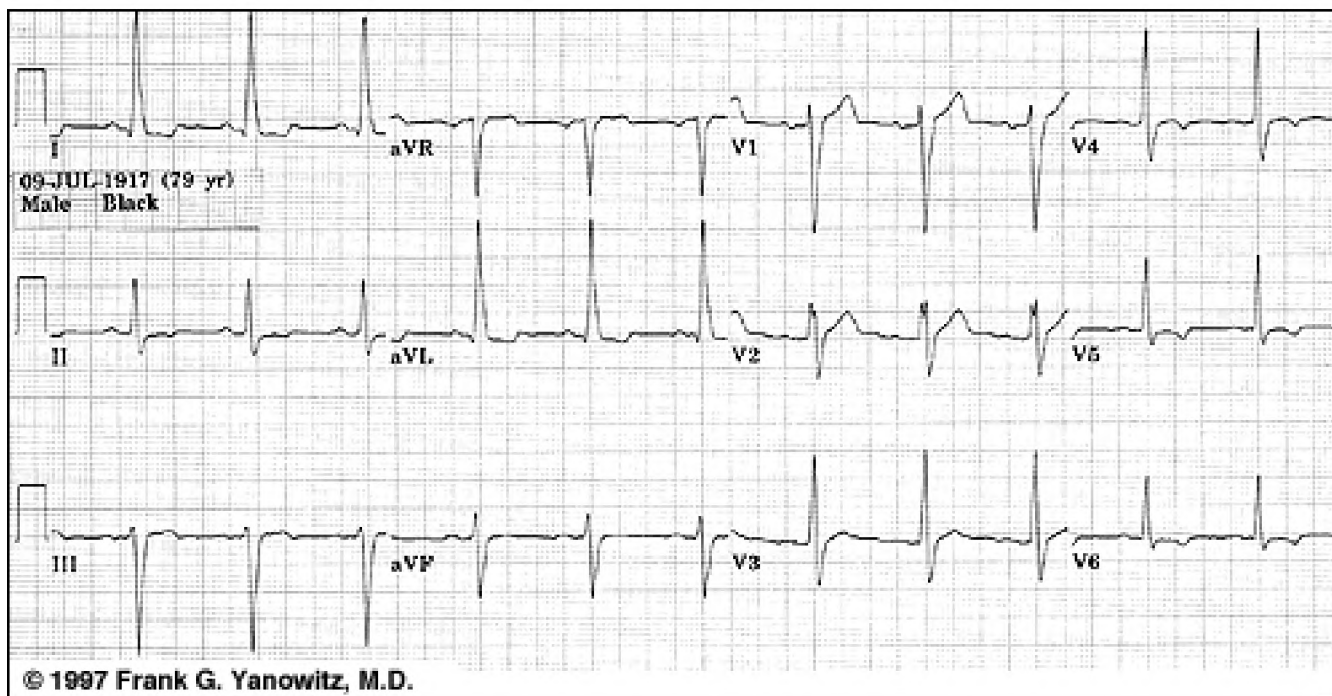
*Frank G. Yanowitz, M.D. copyright 1997*

Note the qR pattern in right precordial leads. This suggests right ventricular pressures greater than left ventricular pressures. The persistent S waves in lateral precordial leads and the RAD are other finding in RVH.



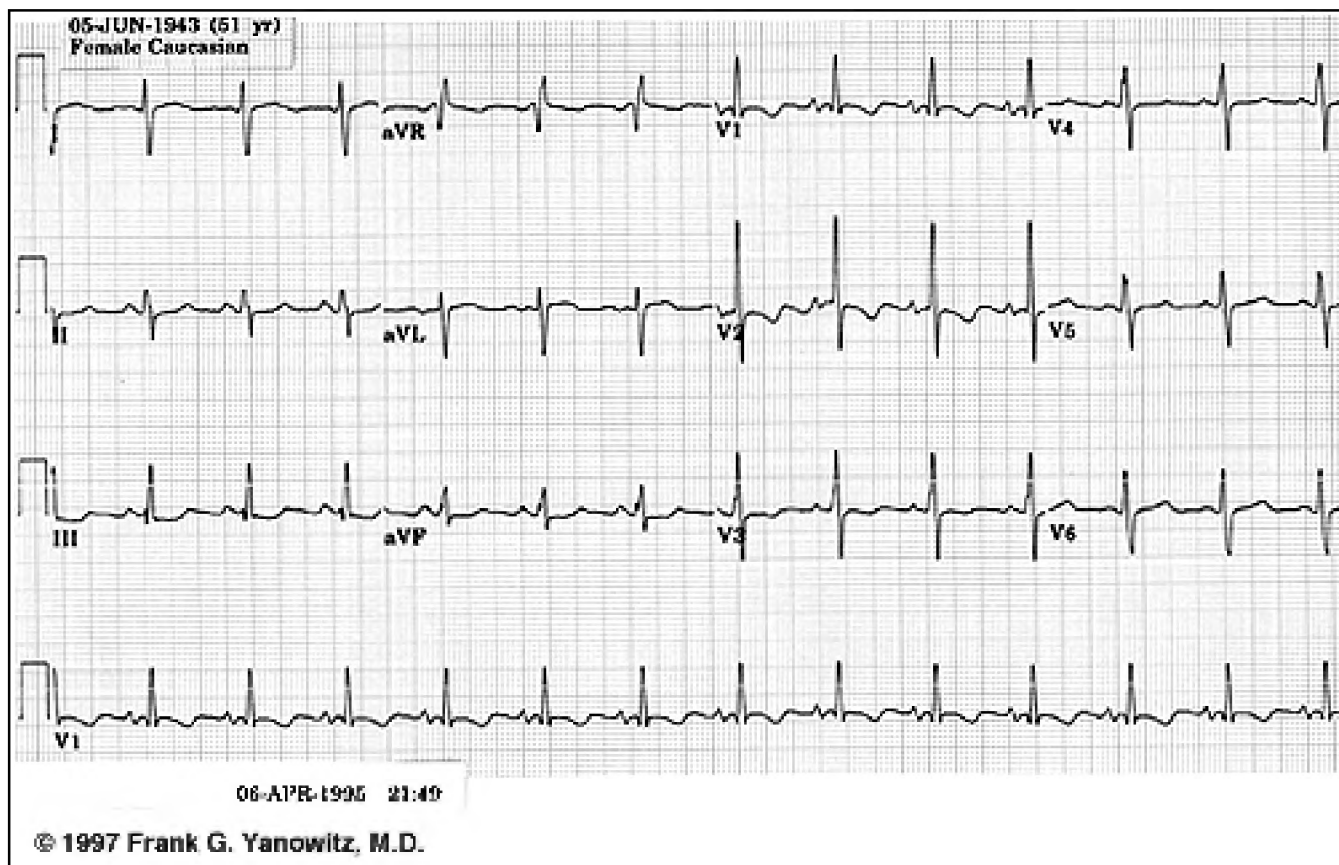
## LVH: Strain pattern + Left Atrial Enlargement-KH

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*Frank G. Yanowitz, M.D. copyright 1997*

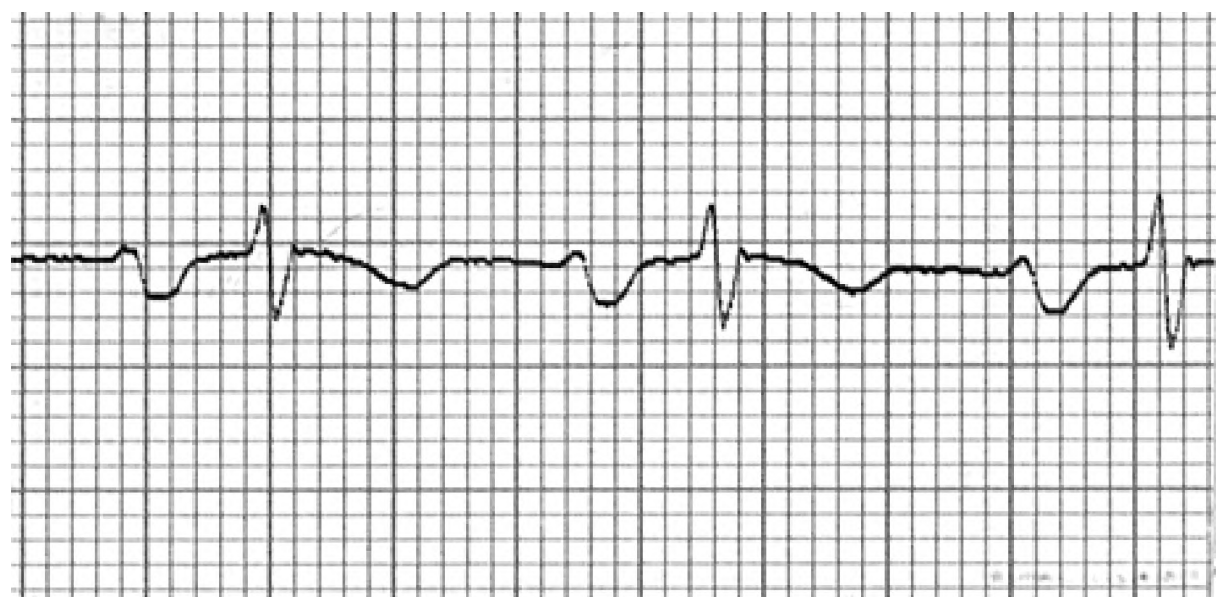




## Severe RVH

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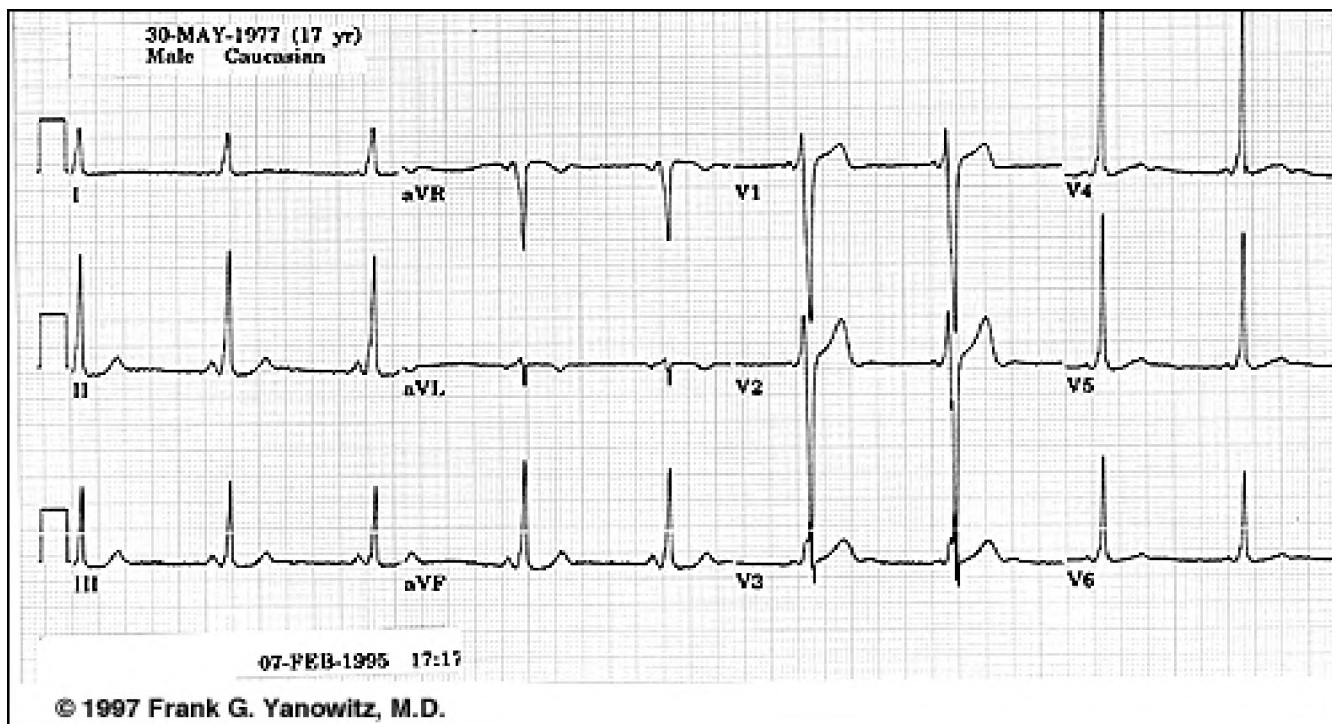
RVH features include the marked right axis deviation (+150 degrees), qR complex in lead V1, R:S ratio in V6 <1, and right precordial lead ST depression.



# Left Atrial Enlargement-KH

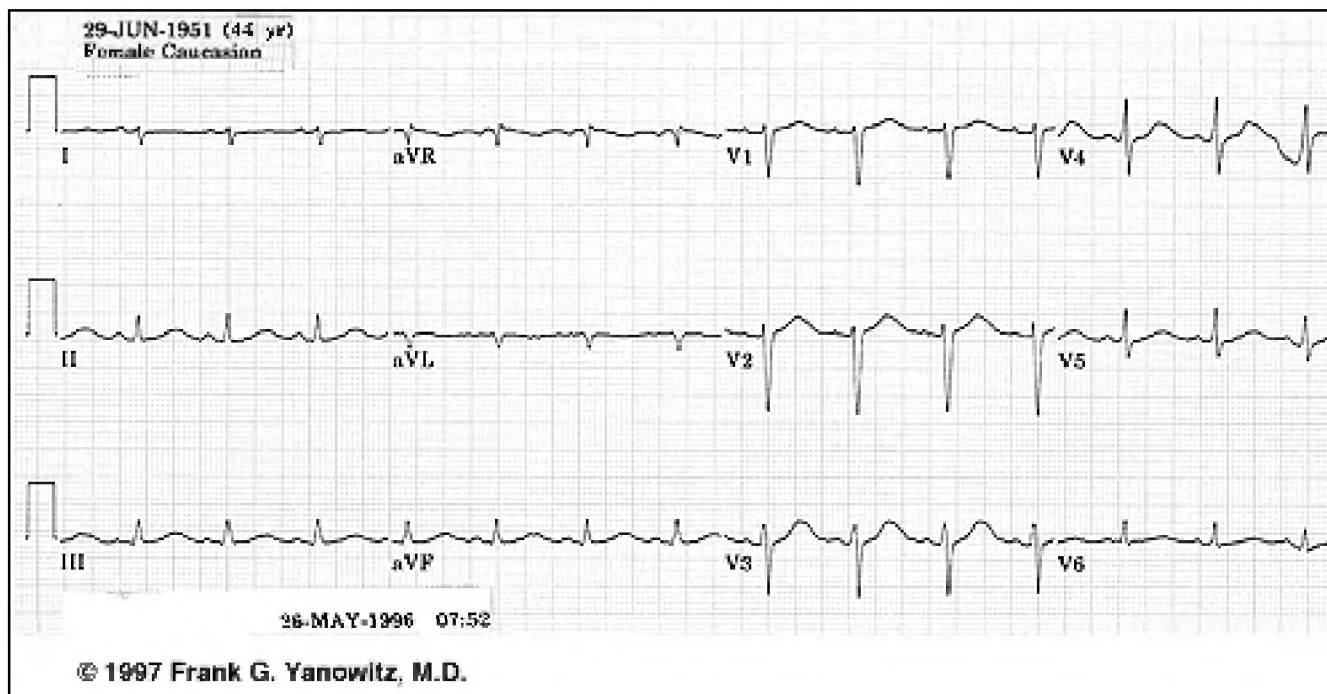
*Frank Yanowitz Copyright 1996*

Left atrial enlargement is illustrated by increased P wave duration in lead II, top ECG, and by the prominent negative P terminal force in lead V1, bottom tracing.



## Long QT Interval and Giant Negative T Waves-KH

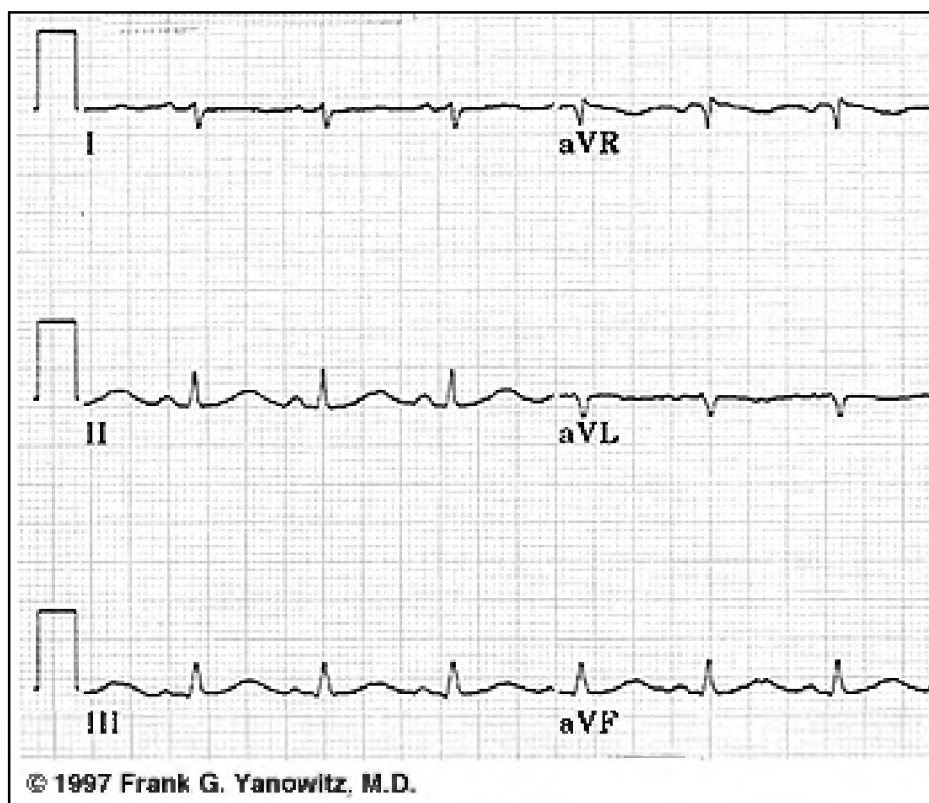
*Frank G. Yanowitz, M.D., copyright 1997*



## Long QT Interval-KH

*Frank G. Yanowitz, M.D.*

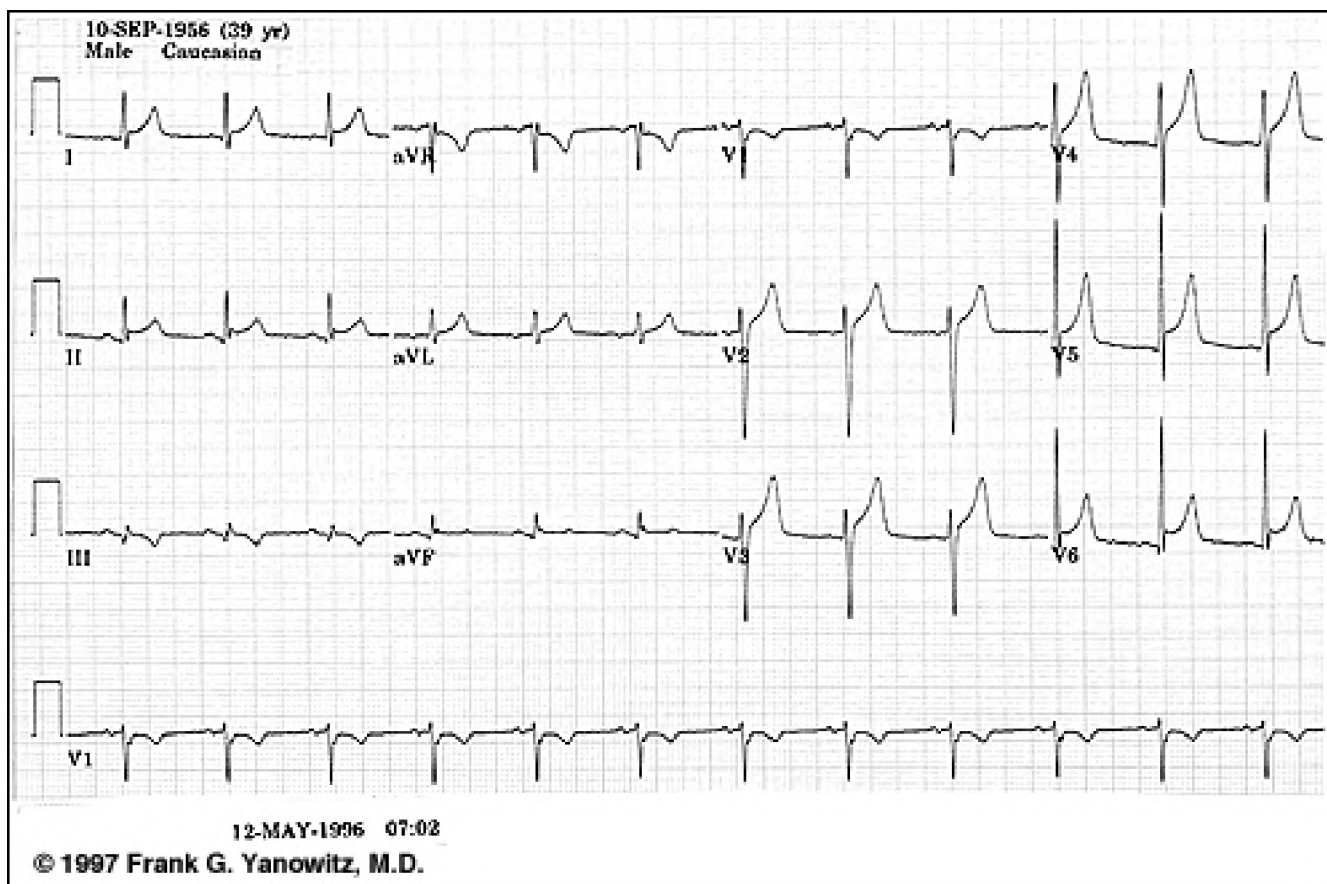
The QT interval duration is greater than 50% of the RR interval, a good indication that it is prolonged in this patient. Although there are many causes for the long QT, patients with this are at risk for malignant ventricular arrhythmias, syncope, and sudden death.



## Long QT Interval-KH

*Frank G. Yanowitz, M.D.*

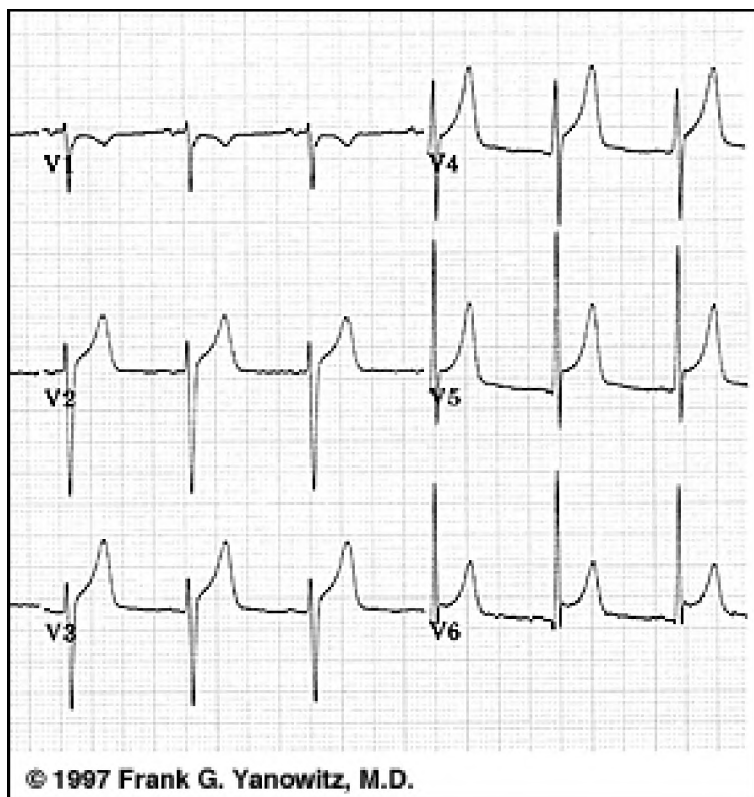




## Normal Variant: Early Repolarization-KH

*Frank G. Yanowitz, M.D.*

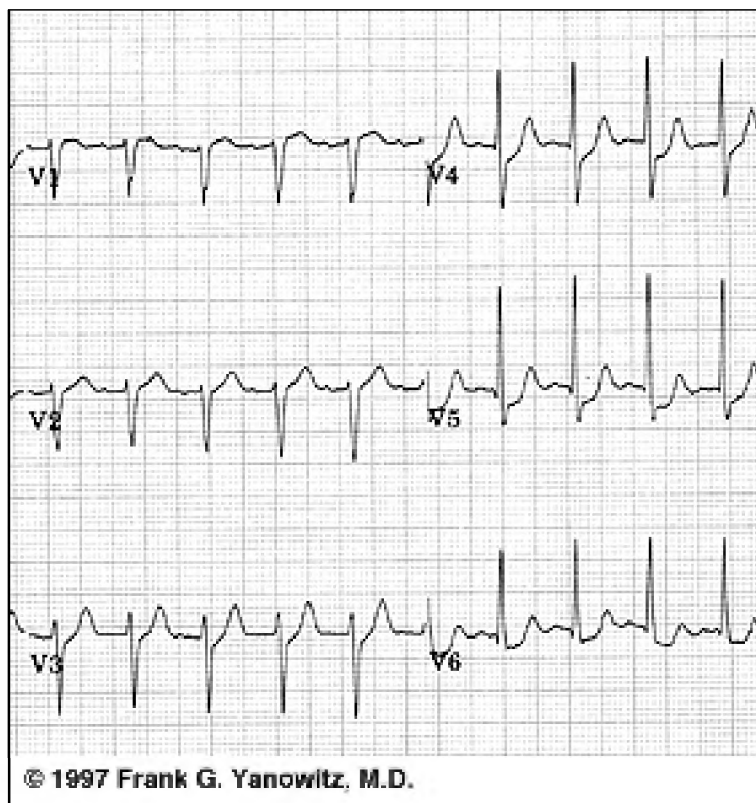
Early repolarization, a misnomer, describes a pattern of localized or diffuse ST segment elevation. This is especially seen in leads with prominent R waves. In this example leads I, II, V5 and V6 illustrate the early repolarization pattern. ST segments usually have a "concave upwards" pattern and take off after a small S-wave is inscribed.



## Normal Variant: Early Repolarization-KH

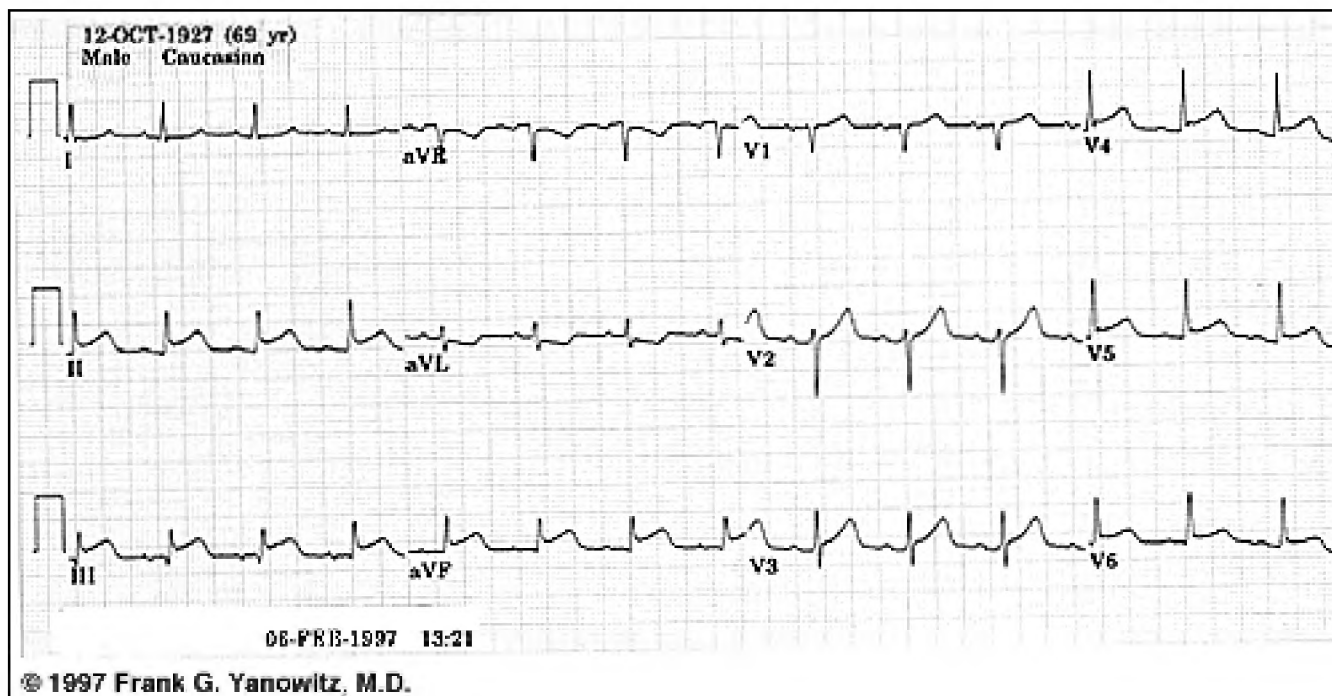
*Frank G. Yanowitz, M.D.*

Early repolarization, a misnomer, describes a pattern of localized or diffuse ST segment elevation. This is especially seen in leads with prominent R waves. In this example leads V5 and V6 illustrate the early repolarization pattern. ST segments usually have a "concave upwards" pattern and take off after a small S-wave is inscribed.



## ST Segment Depression: Precordial Leads-KH

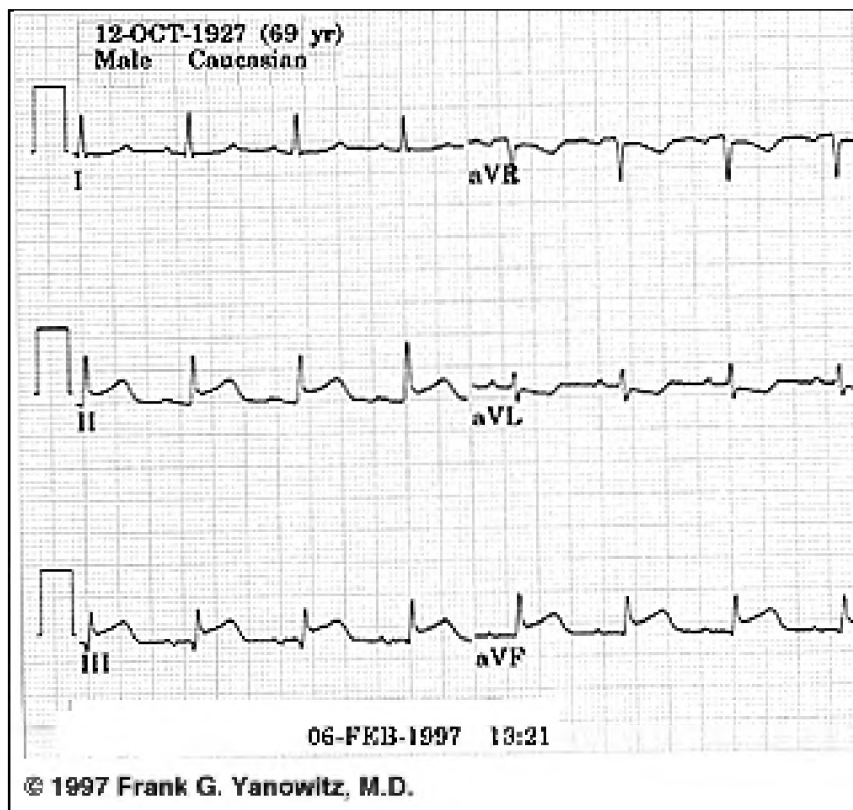
*Frank G. Yanowitz, M.D.*



## Inferolateral ST Segment Elevation-KH

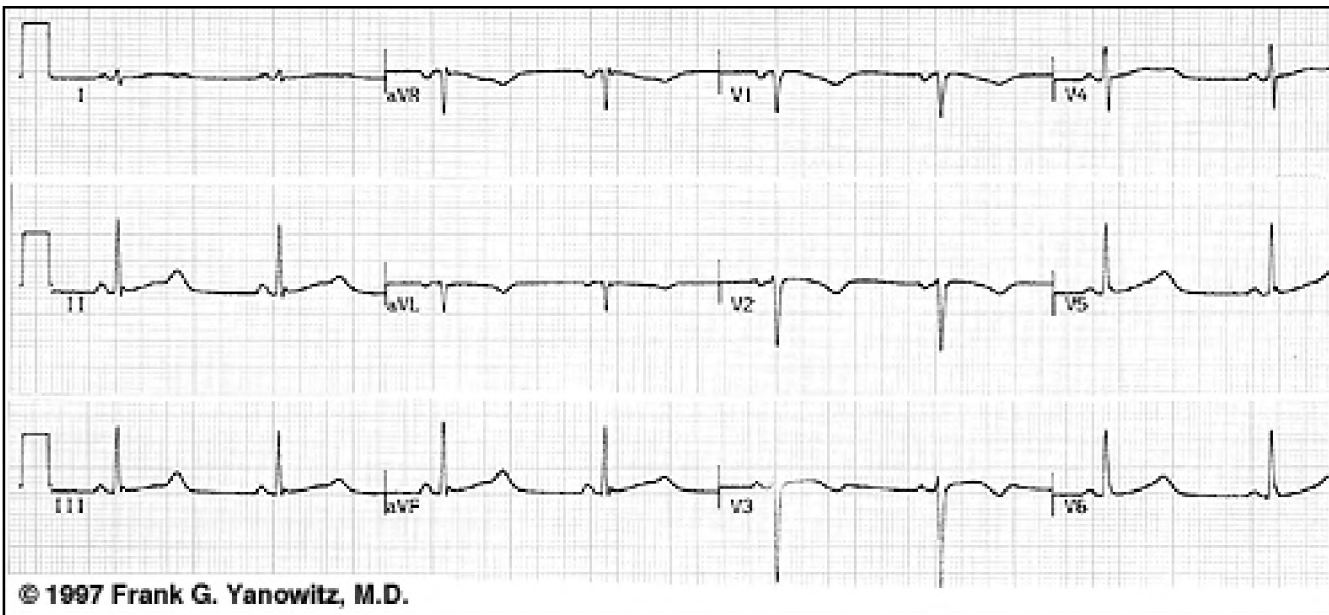
*Frank G. Yanowitz, M.D.*

ST Segment elevation with a straight or convex upwards configuration usually means transmural ischemia (or injury) and is seen in the setting of acute myocardial infarction. This ECG finding may also be seen transiently during coronary artery spasm. Unlike ST depression, ST elevation is often localizing. In this example of inferolateral ST elevation, the culprit artery is often a dominant right coronary artery or dominant left circumflex artery.



## ST Segment Elevation: Frontal Plane Leads-KH

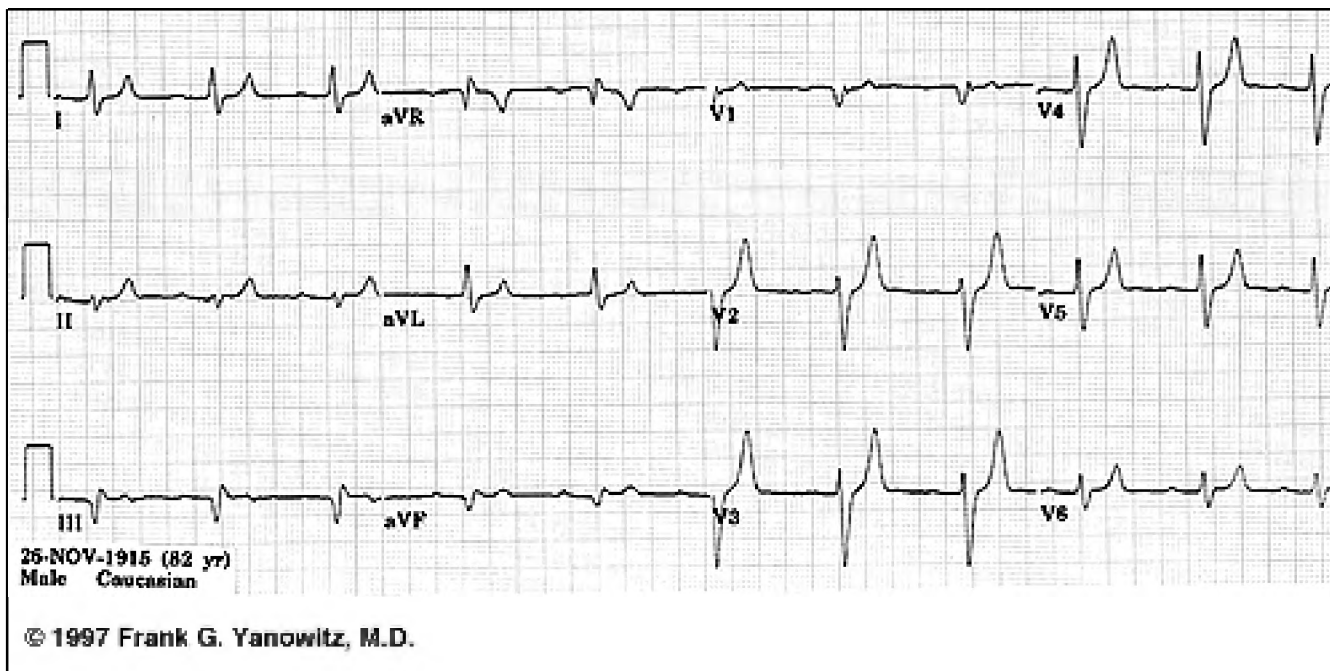
*Frank G. Yanowitz, M.D.*



## Long QT: An ECG Marker For Sudden Cardiac Death-KH

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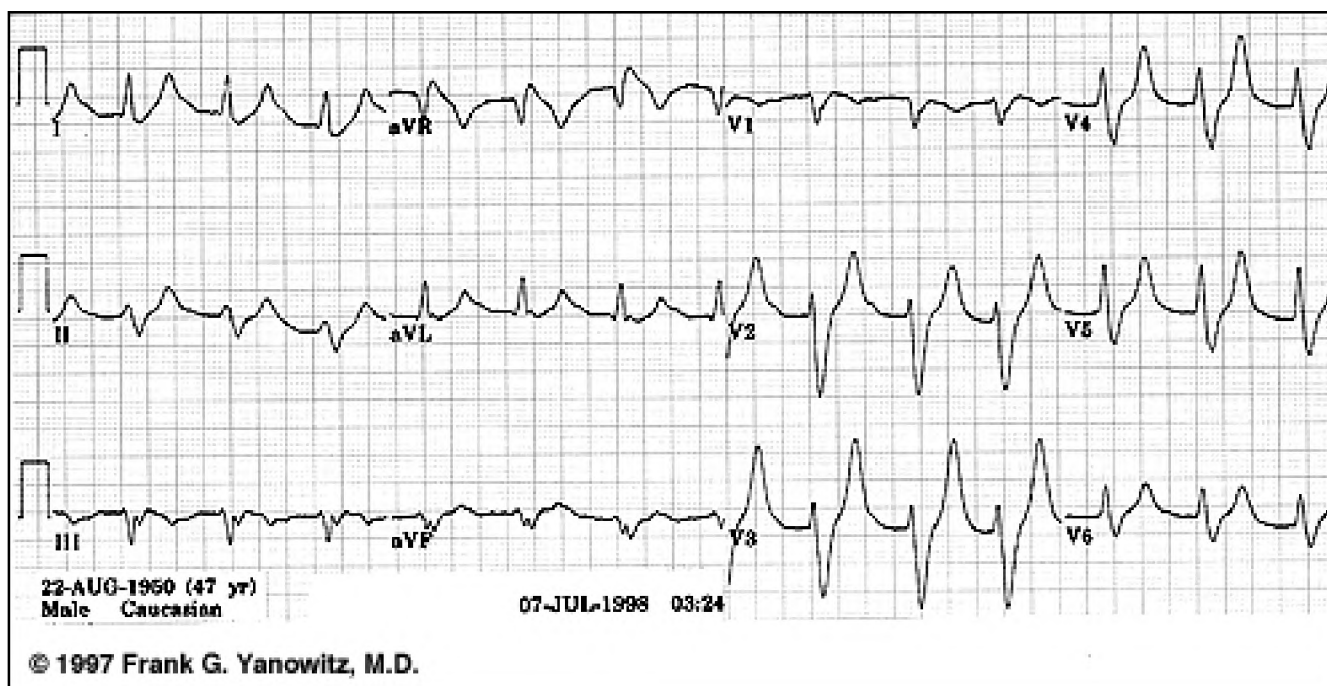




# Hyperkalemia and Old Inferior MI

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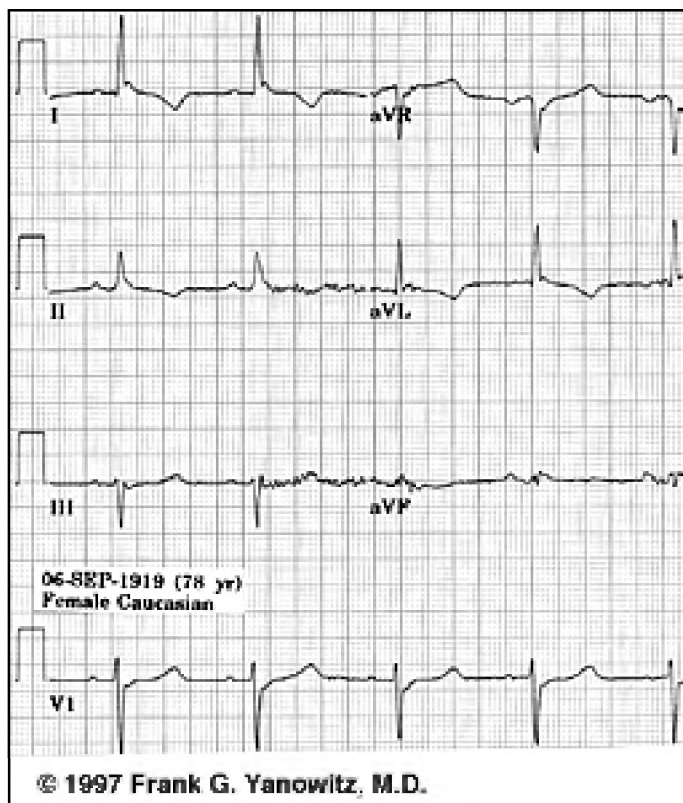
The T waves are tall, peaked and have a narrow base, making them very uncomfortable to sit on! These changes are characteristic of hyperkalemia. The QRS is also slightly widened, another feature of hyperkalemia. Q waves in III and aVF indicate an old inferior MI.



## Advanced Hyperkalemia

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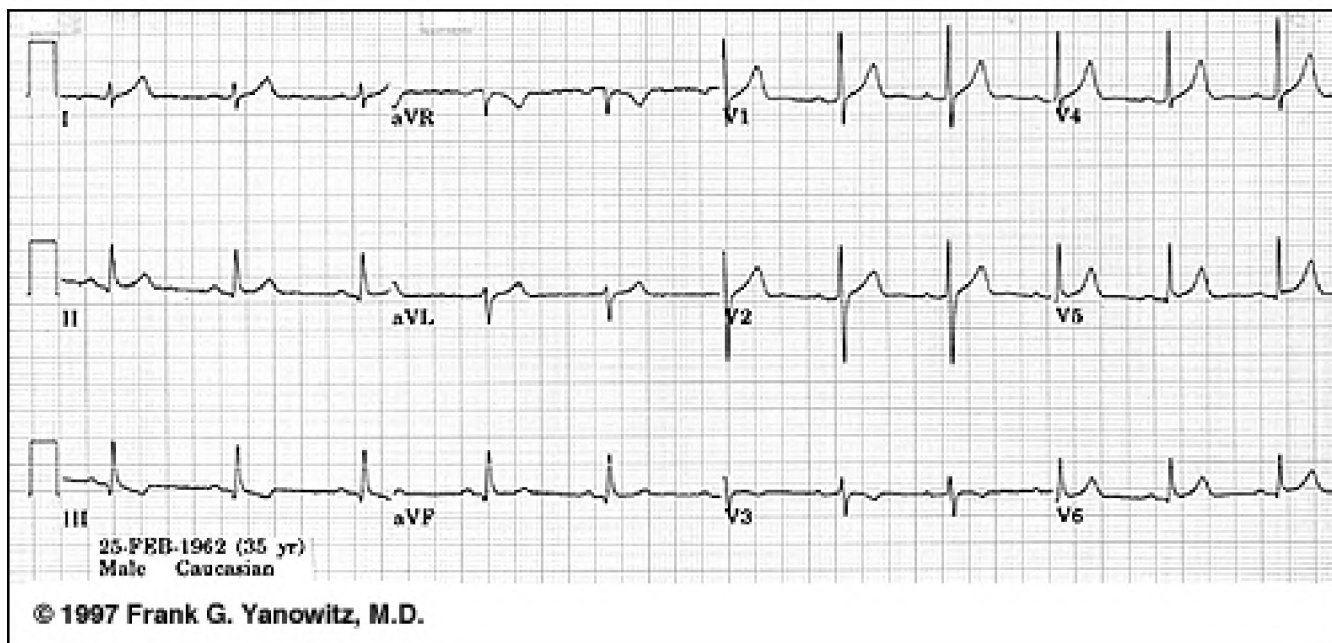
Marked widening of the QRS duration combined with tall, peaked T waves are suggestive of advanced hyperkalemia. Note the absence of P waves, suggesting a junctional rhythm, but in hyperkalemia the atrial muscle may be paralyzed while still in sinus rhythm. The sinus impulse conducts to the AV node through internodal tracts without activating the atrial muscle.



## Hypothermia: J-waves or Osborne Waves

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In hypothermia, a small x-tra wave is seen immediately after the QRS complex (best seen in Lead I in this example). This x-tra wave is called a J-wave, or Osborne wave after the individual who first described it. This wave disappears with warming of body temperature. The mechanism is unknown.

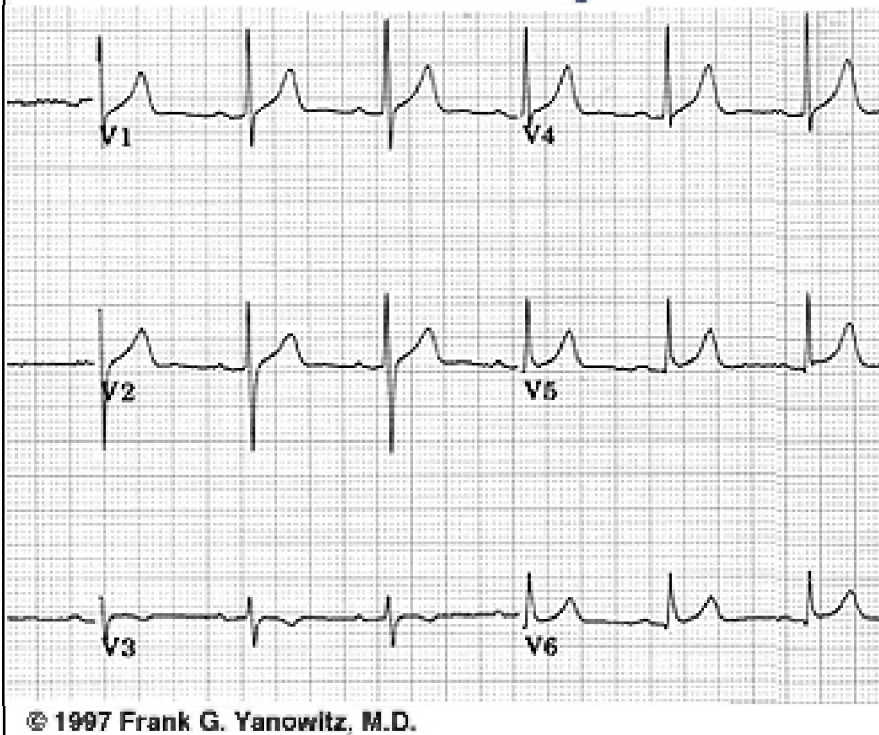


## Lead Error: V1 & V3 are Transposed-KH

*Frank G. Yanowitz, M.D.*

In this normal 12-lead ECG the V1 and V3 chest electrodes are interchanged. Experienced ECG interpreters should be able to spot this lead placement error.

## What's Wrong?

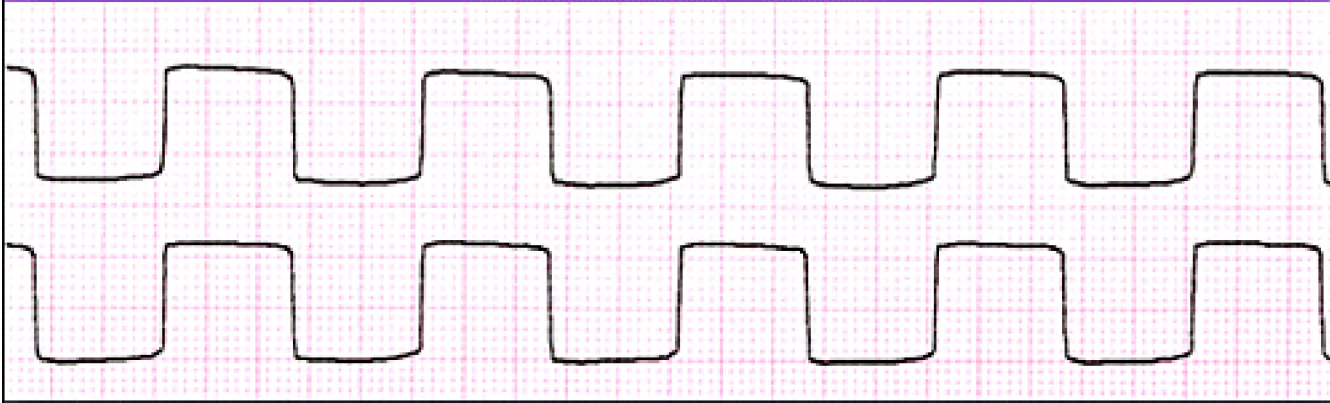


## Lead Error: V1 and V3 are Transposed!-KH

*Frank G. Yanowitz, M.D.*

In the precordial leads the V1 and V3 chest electrodes are interchanged. Experienced ECG interpreters should be able to spot this lead placement error.

## Calibration Pulses



**Deliberate artifact caused to show the interpreter the relationship of the complexes with a known electrical stimulus (standardized procedure).**

## Calibration Signal - Marquette-KH

*Marquette Electronics Copyright 1996*